THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontology; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

1961 Annual Meeting

October 2 - 25, 1961

Convention Cruise—Philadelphia to Bermuda

This is an issue of the
Journal of Periodontology designed to honor
Dr. Harold G. Ray
For cataloging it is designated as the regular October issue

Harold G. Ray, D.D.S.

April 3, 1903—July 17, 1960

Dr. Harold Ray, who had been most active in organizing and presiding over the second workshop on teaching of periodontology, left immediately after the adjournment of this meeting for London, became ill en route to Europe and died July 17, 1960, in Guy's Hospital in London, England.

Dr. Ray, past president of the Academy of Periodontology, was recognized as an excellent teacher and a dedicated worker in dental education. He was active in the teaching of periodontology but always maintained a private practice of dentistry, for the last ten years limiting his activity to perodontics.

On the following pages are some of the many letters received about Dr. Harold Ray.

Dr. Harold Ray, like his teacher Balint Orban, was a dynamic individual with an intense interest in periodontology and a profound desire to teach others of its importance. He chose the activities of the American Academy of Periodontology as a sphere of great influence and worked diligently in the organization toward education in periodontia.

When president of the Academy, he suggested that the Academy sponsor a workshop to study the objectives and methods of undergraduate teaching in periodontology. His proposal was received by the Council with mild enthusiasm and all agreed it would be a good idea worthy of further consideration. This, however, was all that Dr. Ray needed to make his idea a reality. In less than two years he had raised the money to support and publish the results of a workshop on the suggested subject. He selected a committee, organized the entire activity, and prosecuted the workshop in San Francisco. Six months following the workshop, he had edited the proceedings and had the results published in book form for world wide distribution to schools of dentistry to guide them in the teaching of periodontology.

This accomplishment gives some idea of Dr. Ray's directness of purpose, ability for organization, and dedication to periodontia.

The first workshop was so completely successful and productive that Dr. Ray suggested that the Academy sponsor a second one on the teaching of graduate and postgraduate periodontia. The Academy agreed with greater enthusiasm than previously and suggested that Harold and the committee that assisted him with the first workshop prosecute the second one. Again in two years' time, Dr. Ray organized and directed the second workshop which was held in Minneapolis in June of this year. Immediately following the workshop Harold left for Europe to join his family for a well earned vacation and to edit the proceedings of the workshop.

These two workshops will serve as a monument to Harold both in the Academy and in the field of dental education.

Harold Ray was a stimulating teacher who believed that periodontia was the most important phase of dentistry and directed all his energies toward the promoting of his belief. He was an ardent worker in the American Academy of Periodontology where his raspy voice, portly stature, and jovial manner will be sorely missed. Through his untimely death dentistry, and periodontia, in particular, has lost one of its most dynamic, enthusiastic, and forceful teachers. The loss is ours; the reward is his.

Donald Kerr,
University of Michigan
School of Dentistry

A faculty may be thought of as a person—a living, growing, changing individual, the facets of whose character are the contribution of the members who make up the whole person. Some few faculty members make dominant and lasting contributions, and a very few actually shape and influence the destiny of the group. Such a man was Harold Ray.

Those of you who knew him, know what I mean. For those of you who never had the opportunity of experiencing the warmth, the stimulating vitality and intellectual vigor of this man, I could never paint an adequate word picture. Suffice it to say we will eventually appoint a person to succeed Dr. Ray on our staff, but never to replace him.

Willard C. Fleming,
Dean, School of Dentistry,
University of California

The members of Northwestern University Dental School faculty who knew Harold Ray, and many did, were saddened to learn of his death in London on July 17. I arrived in London a day or two after Harold had his heart attack and was confined to Guy's Hospital, and in a conversation with his physician I realized that his condition was very serious.

Harold Ray graduated from Northwestern University in June, 1926, and immediately joined the faculty as a part-time teacher in endodontics and periodontics. I can still recall him as an understanding and helpful teacher. As we all know, his interest in periodontics continued throughout the years. He was not satisfied just to practice in this important field of dentistry, but continued always to take some formal training in subjects basic to a better understanding of periodontology. Even though he separated geographically from Northwestern University, we always had a great regard for him and for what he stood.

George W. Teuscher,
Dean, Northwestern University
Dental School

Dr. Ray was an eloquent speaker and a very effective teacher. He was well liked by his students as best expressed when a senior stated, "it is unfortunate that death should take one of our best teachers from the school." He was a man who was sympathetic with the problems of the undergraduate and graduate student.

Dr. Ray was not only a good teacher of the students, but I too learned from him both as to teaching methods as well as additional information in the field of Periodontology. Only as he is gone am I now beginning to realize what a tremendous task he performed and the odds under which he had to work. Frankly, the balance of the faculty at the school did not share his views in the need for raising the standards of teaching of Periodontology. He gave of his time in constantly combatting the limitations which were imposed upon the department. In addition, he had just completed a presentation to the Academic Planning Committee and obtained the approval for the establishment of a research institute in the field of Periodontology as a part of the Medical Center campus. I need not list his many achievements as a teacher, lecturer, researcher, the father of the Teachers Workshops in Periodontology, member of the American Board of Periodontology, an influential man in raising the standards of dentistry and periodontia in the service and in lay dentistry.

Destiny has ordained that few men should live and contribute as much as did Harold Ray. No greater tribute can be paid anyone in his obituary than to say that the world is better off for his having lived. I consider it a signal honor to say that he was my friend.

Perry A. Ratcliff,
School of Dentistry,
University of California

I was shocked to hear of the untimely death of Dr. Harold Ray. All who have had the privilege of knowing him are aware of the great loss this is to the dental profession.

With his enthusiasm and capacity for organizational effort, Dr. Ray made great contributions to the field of dental education and the specialty of Periodontology. He will long be remembered for his contributions to the progress of dentistry as a teacher and scientist.

Joseph L. Bernier,
Major General, D. C.,
Assistant Surgeon General
and Chief of the Dental Corps

The passing of Harold Ray leaves another void in the roster of Academy membership and in the field of periodontia. He was a man of boundless energy and capacity to get things done. His enthusiasm and dedication to the cause of better teaching in periodontia were an inspiration to all who knew him. Those of us who had been on his staff at the University of California Dental School had great respect for his teaching ability and his talent for organization. Whether in the affairs of the Academy, at the School or meetings of State and Local Dental Societies, Harold was untiring in his efforts to do the very best that he was capable of doing. He set a pattern that will be difficult for those who follow in his foosteps. One of the highest ideals in life is Service—Harold had that ideal.

Dickson G. Bell, D.D.S.,
San Francisco 8, California

The news of Harold Ray's death produced in those who knew him best a reaction of shocking disbelief, so vital was his presence during his lifetime. One suspects that as the months roll by, there will be further reluctant realizations of our loss as we enter situations in which we were accustomed to having his help. His views on curriculum development and long range planning for the University of California are fortunately set down in writing, but to take a selfish view, we would be so much better off if we could have him with us to face the future which interested him so much.

Wendell L. Wylie,
Associate Dean, School of Dentistry,
University of California

The periodontal community of San Francisco lost a great leader in the passing of Harold G. Ray. His lofty ideals of the role of periodontics in organized dentistry coupled with his creative genius and enthusiasm for hard work left a lasting imprint in the minds and hearts of all of us, who were privileged to work with him.

Alvin C. Hileman, D.M.D., M.S.,
Chairman, Department of Periodontics,
College of Physicians and Surgeons
of San Francisco

And, around us Death's inexorable hand
Draws the dark curtain close; undrawn no more
(The complaint, Night 6)

If ever, these words of poet *Edward Joung* have found these days a frightening implementation within the ranks of world's leading parodontologists. After the sudden passing away of B. Orban and the dramatic death of K. Häupl on June 29, there reached us on the 17th July from London the sad message that *Harold G. Ray* of Palo Alto, California, has succumbed there of a heart failure.

Harold G. Ray, a full time professor and chairman of the Department of Periodontology at the University of California School of Dentistry, San Francisco had just arrived in Europe where he was to spend his sabbatical leave which he had meticulously mapped-out to serving and enriching his chosen profession. He was scheduled on the programmes of the Congress of the American Dentists of Europe in Edinburgh, the Centennial Celebration of the Swedish Dental Association in Stockholm, and the Congress of Arpa International in Vienna.

But coming to Europe Harold Ray, the type of American scientist of the newer generation, was fully aware that such a visit has a reciprocal value. "Do ut des" (I give, thou give me) was his conviction; to this he planned to make his headquarters in Düsseldorf at the Westdeutsche Kieferklinik of K. Häupl in order to learn the many beneficial aspects of functional therapy. He planned to visit with many other universities; to come to Geneva, the headquarters of ARPA and WHO, to learning about the international aspects of his specialty. He availed himself of the introduction to many health ministries including those of Poland and USSR.

Dr. H. G. Ray was born in Chicago in 1902. He was graduated from Northwestern University College of Dentistry in 1926 and taught on the staff there in dental pathology until 1933. His keen interest into the problems of periodontia was much fostered by the personal contact with B. Orban. At the age of 45, giving up a flourishing private practise, he took up graduate studies at the University of Illinois. In 1949 he was given a Master of Science degree in histology after having submitted in partial fulfillment of the requirements a thesis entitled "The gingival structures in diabetes mellitus" (J. Period. 21:85, 1950). This proved to become a turning point in his life. His inner vocation for learning and teaching determined him to accept the offer of Dr. H. Becks to join the staff of this outstanding leader in Oral Biology at the University of California. Dean Fleming, himself today one of America's greatest dental educators, soon recognized the rare qualities of this combined researcher and practitioner and entrusted him the newly created chair of periodontology.

The accomplishments of H. Ray in the field of periodontology are impressive. His clear, systematic manner of thinking coupled with his broad experience and knowledge made him an excellent teacher. His courses were highly appreciated by his undergraduate and postgraduate students. Beyond the borders of the States he was a much sought-after speaker at meetings and conventions. The 38th Annual Meeting of the American Academy of Periodontology installed him president specifically honoring him for his services rendered to the science and dental specialty of periodontology.

For the advancement of his dental specialty he clearly recognized two avenues: more research, better teaching. In pursuing this objective on a local, as well as national basis he made the gigantic efforts that finally broke his physical forces. To foster periodontal research he succeeded just before death to establish a research center at his own school. With his *Workshop for Teachers in Periodontology* sponsored by the American Academy of Periodontology he gave the profession definite guidance how to incorporate, to enhance and to validate the teaching of periodontology as a means to better and more comprehensive dental health service. A recent expert Committee of the World Health Organization has recognized the value of this contribution and adopted the principles therein established.

So it can be truly said that Dr. Ray's greatest contribution to periodontia was in the field of education. The influence of his strides is world-wide. Humanity is enriched by his work and life. Harold G. Ray was an example of a very fine and educated personality; self-controlled in any situation, just in every decision, rich and warm in his affections he lived up to the Golden Rule. When an intense desire to serve humanity is accompanied by an able, unpretentious, genial personality the result can mean a great man. Such was Harold G. Ray. He never can be replaced in the affection of his students and friends.

The sympathy of all is extended to his beloved wife the understanding companion and counsellor in his great strides and victories.

Louis J. Baume,
President SSO

The specialty of periodontics, the American Academy of Periodontology, and the American Board of Periodontology have suffered a great loss in the death of Harold G. Ray. His whole professional life has been dedicated to practice, pedagogy, and journalism in this field. His contributions are a monument to his efforts, and he will always be remembered in the highest esteem by his close friends and his legion of professional friends.

Clarke E. Chamberlain, D.D.S.

It was with deep regret to hear of the untimely death of our colleague Dr. Harold Ray. His contributions to the field of periodontology are well-known by the dental profession. By his diligence and his organizational ability he helped raise periodontology to the high level it now enjoys. A recent example was the outstanding manner he organized and conducted the recent Workshop for Teachers of Periodontology.

His unselfish devotion to the academy and other organizations will long be remembered. The name Harold Ray will always be associated with an individual who placed his profession and friendships foremost.

Henry M. Swenson, D.D.S.

The Effect of Anticonvulsant Drugs Upon the Gingiva—A Series of Analyses of 1048 Patients

I

by HAROLD J. PANUSKA,* ROBERT J. GORLIN,* JACOB E. BEARMAN,** AND DAVID F. MITCHELL***

SINCE the introduction of Dilantin (Dilantin sodium, diphenyl hydantoinate, Epanutin, diphenyl hydantoin) as an anticonvulsant drug in 1936 by Merritt and Putnam,[24] there have been many reports published about its well known side effects, including gingival hyperplasia. Because of the long use of Dilantin at the State School and Hospital for Epileptics in Cambridge, Minnesota, a survey was made to attempt to correlate many of these reported side reactions.

DILANTIN GINGIVAL HYPERPLASIA—REVIEW OF THE LITERATURE

Gingival hyperplasia has a reported incidence ranging from 3 to 70 per cent in patients under treatment. This wide range is probably due to the following: (a) ill-defined criteria of hyperplasia have been used; (b) much of the information was gathered by persons other than dentists, and (c) no consideration was made of the amount of time patients were taking the drug. Table I demonstrates the percentage incidence reported by various workers since 1939. Despite the number of side reactions reported, none are so serious as to warrant discontinuation of the use of the drug. Dilantin enjoys a position of real importance today as an anticonvulsant, and as yet cannot be replaced by any other drug. In 1942, Prudhomme[30] reported that Dilantin was 62 per cent more effective than previous medication over the same period of time in the control of seizures. More recently, Bieter and Wright[4] report 60 to 85 per cent improvement.

The mode of action of Dilantin suggested by Aird[1] is due to its effect upon the permeability of the blood-cerebrospinal fluid barrier. He further suggested that the lowered permeability of the cell membranes is associated with epilepsy. Staple[36] suggested a relationship to the adrenals which he indicated were heavier in epileptics and that their cholesterol content was greater. He suggested that Dilantin causes hypertrophy of the adrenal glands, also proposing that gingival hyperplasia is an exaggerated connective tissue response to local oral stimuli in patients with deranged adrenocortical functions. He also stated that salicylates stimulate adreno-cortical function and counteract the anticonvulsant effects of Dilantin sodium.

Characteristic oral manifestations were described by Orban and Wentz[28] and Thoma.[41] The gingiva showed marked hyperplastic enlargement beginning with overgrowth of the interdental papillae, involving finally the entire gingival margin. The teeth appear partially or at times even wholly submerged in the excess tissue. The condition may be generalized or localized, appearing lobulated when isolated areas are involved. These lobules may form clefts by juxtaposition with each other causing the patient to appear as if he had a mouth full of strawberries. There is no bleeding or increased sensitivity of the gingival tissue except in cases of superimposed inflammation caused by poor oral hygiene. Hyperplasia has been noticed in other areas of the mouth, e.g., two papillomatous

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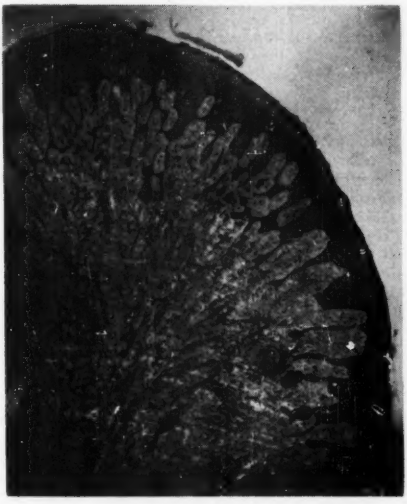

Fig. 1. A low power photomicrograph illustrating the fibrous and epithelial hyperplasia associated with Dilantin.

palatal lesions were found which recurred after excision.[38] Other investigators reported lack of hyperplasia in edentulous areas of the mouth.[27, 42, 13]

There are no significant detrimental effects of the hyperplasia except for the difficulty it causes in eating and cosmetic deformity. Swinehart[40] noted progressive malocclusion in a particularly long-standing case. Pseudo-pocket formation makes oral hygiene difficult. Pockets are formed around the clinical crown, the normal level of the gingival attachment usually being undisturbed. It is a positive type of pocket, in contrast to a negative destructive type of periodontal pocket.

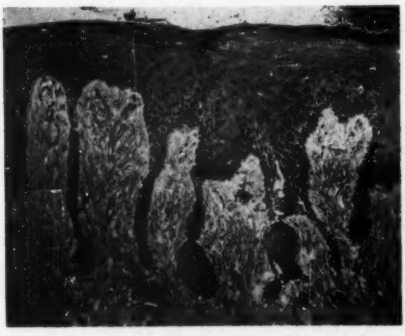

Fig. 2. A higher power view illustrating the epithelial activity including discrete pearl formation. The inflammatory reaction is not remarkable.

The hyperplastic reaction occurs in about three months according to some;[11, 28, 37] however, there seems to be no correlation between the amount of hyperplasia, the length of time the drug is taken, or the dosage, according to others.[13, 42, 38, 27, 37] Local irritation has been suggested by many authors[13, 42, 18] as a complimentary factor in gingival hyperplasia, but others have reported no association with poor oral hygiene.[27, 18] Siegmund advanced a unique theory in his studies. He reported a case of hyperplasia in a patient who received no Dilantin but was suffering from epilepsy. This he explained was due to local inflammation causing gingivitis and leading to fibrous hyperplasia peculiarly related to the brain damage. He went on to explain that the Dilantin only compliments this basic process.[34] Millhon and Osterberg suggested that the intrinsic causation was due to the drug itself.[27] Ishikawa, reporting six cases of unilateral hyperplasia, noted that most of these patients were suffering from a unilateral disorder of the central nervous system, such as hemiplegia or Jacksonian epilepsy. He believed this demonstrated that the gingivae are under the control of the central nervous system.[20]

Certain areas of the mouth have been reported to have a greater incidence of hyperplasia than others. Several authors have reported a greater incidence in the upper anterior region.[16, 27, 13] Others suggested that the areas near orifices of salivary ducts have a greater incidence.[31, 6] Neither group offered any evidence to support these beliefs. Stern *et al* attempted to show the effect of Dilantin upon salivary secretion but no appreciable effect was observed.[38]

Although no correlation between the dosage of Dilantin and the amount of hyperplasia was noted, there are several

cases reported where a quick increase in the dosage, in attempts to control seizures, has caused or has been associated with an increase in actual hyperplasia, or has been the cause of primary precipitation of hyperplasia.[13] Two cases of this type have been observed by the authors. Subsequent reduction of the dosage did not reduce the hyperplasia but maintained it in status quo.

It was suggested that ascorbic acid deficiency hyperplasia resembled Dilantin hyperplasia,[22] but after careful investigation, Millhon and Osterberg,[27] and Ziskin *et al*[42] decided that the two are distinct entities and not associated. Merritt and Foster concluded that gingival hyperplasia developing in patients under Dilantin therapy was not related to the ascorbic acid content of the plasma nor to ascorbic acid utilization.[26] It appears now that this has been well substantiated.

Staple has suggested that Dilantin may inactivate circulating hormones or potentiate them.[36] Ziskin *et al* studied the relationship of Dilantin to estrogenic compounds and concluded that there is no relationship.[42] Ishiwaka reported several cases of hormonal imbalance evidenced by early development of secondary sex characteristics and hirsutism, which he attributed to the use of Dilantin.[20] Reyes[31] and Esterberg and White[13] reported the existence of excessive body hair among epileptics and suggested that this may have been caused by Dilantin.

The relation of age to hyperplasia has been reported. Patients ranged in age from 3 to 69 years. The hyperplasia began at 5 years and the incidence rose sharply from 9 to 12, then slowly to age 24 falling off quickly at age 35.[13] Ishikawa reported the highest incidence between ages 10 and 19, being especially pronounced at age 15.[20] The youngest patient observed in our series was 3 years old with a grade three hyperplasia.

There appears to be no sex or racial correlation with the occurrence of hyperplasia around the upper and lower teeth.[11, 38, 37] Allergy or hypersensitivity has been implicated by Reyes,[31] Bernier[3] and Thoma.[41] They indicated that the relative infrequency, the inconsistency, and the similarity of the side reactions, i.e., dizziness, rashes, and headache, are good reasons to suspect allergy or hypersensitivity. Gaillard[17] reported two cases of successful antihistaminic therapy for hyperplasia due to Dilantin, but recent reports fail to substantiate this.[43]

TABLE I

Summary of Authors Reporting Prevalence of Hyperplasia

Author	*Number of cases*	*% Showing change*
Berg and Perlman[2]	14	57
Esterberg and White[13]	244	54
Fetterman[14]	28	25
Frankell[15]	48	62
Glickman and Lavitus[18]	55	18
Kimbal[23]	152	57
Merritt and Putnam[24]	332	3
Millhon and Osterberg[27]	17	35
Pinsky[29]	—	60
Stern, Eisenbud and Klatell[35]	50	52
Swinehart[40]	—	50
Ziskin, Stowe and Zegarelli[42]	55	18
Ishikawa[20]	887	56
Dummett[11]	123	38
Spira[45]	52	70
Panuska, Gorlin, Bearman and Mitchell	546	32

Histologically, (Figs. 1-2) Dilantin hyperplasia was well described by several investigators.[11, 13, 41, 42] There is acanthosis of the epithelium with occasional pearl formation. An increase in the collagen fibers of the submucosa is prominent. Some bone metaplasia (believed not to be true bone but metaplastic bone) has been described. Secondary inflammatory elements may be encountered. Increased epithelial cell mitotic activity has been reported.[13, 42]

ANIMAL EXPERIMENTATION

This survey was performed after several attempts by one of the authors (H.J.P.) to produce gingival hyperplasia in laboratory animals. Twelve rats and 12 hamsters were fed a soft, high carbohydrate diet. Daily doses of Dilantin, individually mixed for

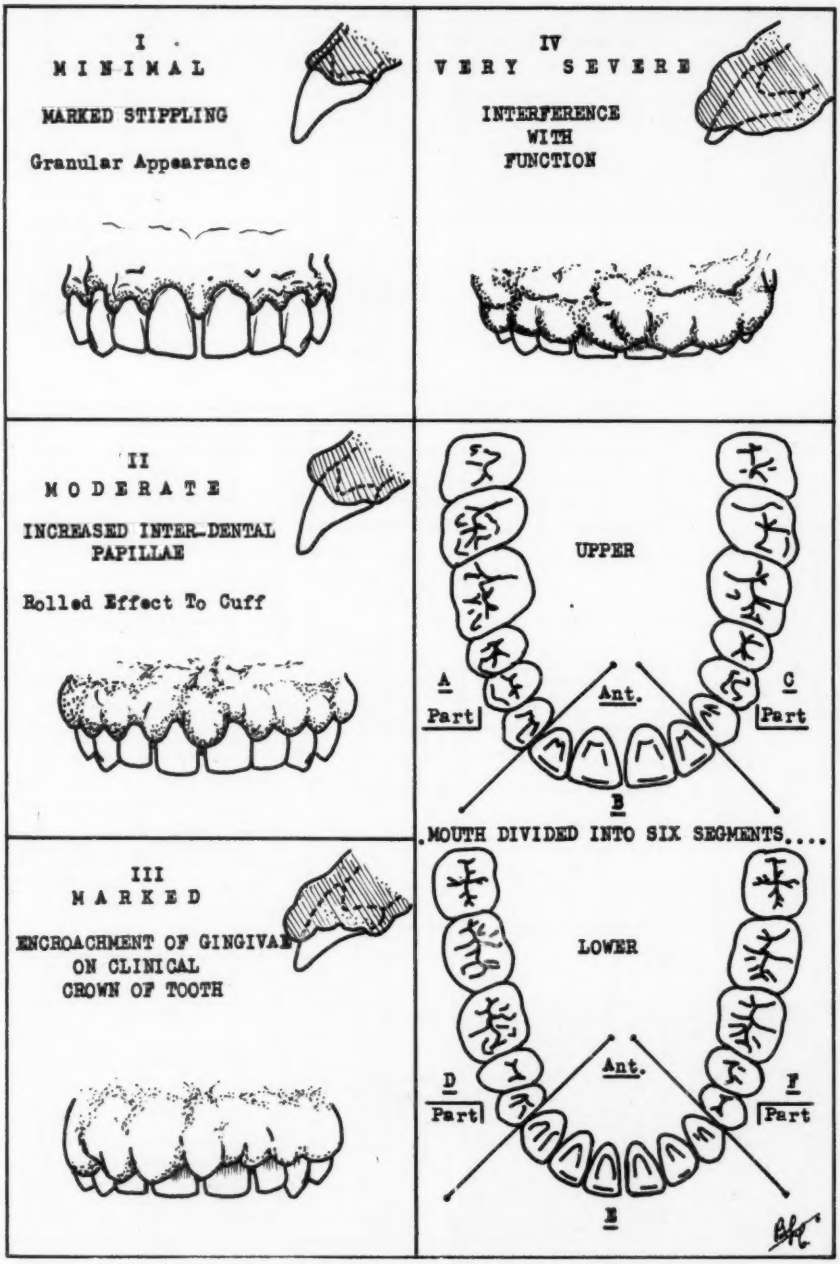

Fig. 3. The divisions of the mouth studied and the degrees of hyperplasia recorded.

University of Minnesota
School of Dentistry
Dept. of Oral Pathology

Number 1 2 3 4 5 6

Survey Conducted at _____ Date ____19__ Recorder _____
Code 7

Basic Information			
Age			8 9
Sex	M 1 / F 2		10
Race	W 1 / N 2 / A 3		11
Years in Hosp.			12 13
I.Q.			14 15 16

Medication			
	X	1	17
	N	2	18
Years of Medication			19

Drugs used at Present in			
DM1 Therapy	Y 1 / N 2		20
DM1antin			21
Phenoberb			22
Mesantpin			23
Bromide			24
Drug 1			25
Drug 2			26
Drug 3			27

Epilepsy Control		28
Type of Seizure		29

Missing Teeth By Area		
Ant Up		30
Ant Dwn		31
Post		32
Post		33
Post		34
Post		35

Fracture		
Upper Ant 1-6		36
Lower Ant 1-6		37

Degree of Hyperplasia		
Ant Up		38
Ant Dwn.		39
post		40
post		41
post		42
post		43

Oral Hygiene		44
Prophylaxis		45
Decay		46 47
Missing		48 49
Filled		50 51
Excess Body Hair		52
Photo		53

Table II

each animal to assure complete intake, were given for one year without success. Because of failure with these animals, an attempt was made to repeat the work of King, who reported the induction of Dilantin gingival hyperplasia using 12 female ferrets.[23] The same number of animals were maintained similarly and were sacrificed at the end of the allotted time but no hyperplasia was observed. Swenson[39] (rats), Shafer[33] (rats) and Ennever and Pendo[12] (Swiss mice) all reported unsuccessful results with laboratory animals. Ishikawa[20] (cats) and King[23] (ferrets) authored the only two successful reports of experimental hyperplasia found in the literature.

Fig. 4. No gingival hyperplasia recorded for this comparatively normal mouth of an epileptic receiving Dilantin.

PURPOSE AND LIMITATIONS OF THE SURVEY

The purpose of this survey was to evaluate the oral condition of the patients at Cambridge State School and Hospital in Cambridge, Minnesota, with particular attention to the influences of the anticonvulsant drugs upon the oral mucosa, particularly the gingiva. A topographical study of the hyperplasia in six different areas of the mouth was done to examine the possibility of increased prevalence in particular areas. Our classification of oral hygiene and hyperplasia can, with minimal practice, be reproduced by others. With new anticonvulsant drugs becoming available almost overnight, a study of several of the newer ones and their effects in a "blind method" was planned. Many patients receive combination therapy involving as many as five drugs. Since multiple drug therapy would interfere with simple analysis, it was decided to use this to best advantage and study, as well, the increase or diminution of gingival hyperplasia when these drugs were used in combination with Dilantin.

In a survey of this type where large numbers of patients were reviewed, each presenting a large amount of gatherable information, we could only hope to use an epidemiological approach and establish as many empiric relationships as possible. These relationships were, of necessity, gross in nature. There are other limitations to this study. Many of the data have been subjected to the error of individual variation. Although an objective appraisal was made, subjective influences could not be entirely eliminated; however, every attempt was made to minimize these. We found that all the information sought was not available to us, and some records were incomplete.

SURVEY METHODS AND TECHNIQUES

Oral examinations of 1048 patients were performed in the cottage units by one of the authors (H.J.P.) and the resident dentist, with the help of a recording secretary. The patients were seated in a straight-backed chair equipped with a portable headrest. An oral surgery headlight and torque blades were used.

The following information was recorded on special mimeographed forms prepared for this survey as illustrated in Table II.

Fig. 5. (Stage I) Minimal hyperplasia characterized by an increase in density and size of the gingivae especially in the lower anterior region.

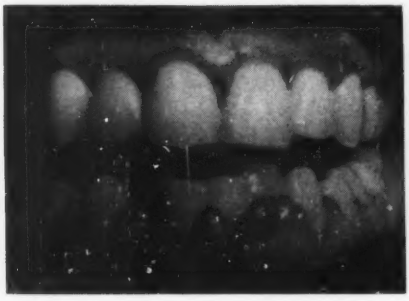

Fig. 6. (Stage II) Moderate gingival hyperplasia especially in the lower anterior region.

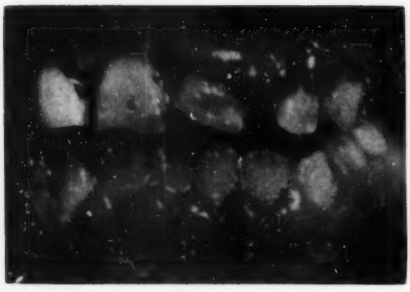

Fig. 7. (Stage II) Moderate hyperplasia, more generalized.

The form was arranged to facilitate transfer of the information to International Business Machine cards No. 5081 to facilitate abstracting of the material. The first two items recorded indicated either the cottage unit number or the patient's hospital ward. The next four figures were the patient's hospital record number for identification purposes. The age and sex of each patient was recorded with his race which was classified as (1) white, (2) Negro, or (3) other. The number of years the patient spent in the hospital was transferred from the dental record where the admission dates were noted. The Intelligence Quotient was taken from the hospital population record.

The daily medication for each patient was taken from the current medication charts in the individual cottages. All drugs and their dosages were recorded to the nearest *full grain per day*. A special column was left for each of the drugs in the following order: Dilantin, phenobarbital, Mesantoin, bromide, Mebaral, Hibicon, and a miscellaneous column for Tridione and others. New combination drugs like Phelantin (½ gr. phenobarbital—1½ gr. of Dilantin) and Mebaroin (1½ gr. of Mebaral—1 gr. Dilantin) were separated into their individual components before being tabulated.

An estimate by the cottage aide of seizure control for the patient was noted. These estimates were rated: (1) good; (2) fair; (3) poor. Electroencephalographic (EEG) tracings and their interpretations were available for most of the patients and they were recorded in case the information might be needed at a later date. The EEG classifications that were used by the institution were: (0) normal, (1) grand mal, (2) petit mal, (3) grand and petit mal, (4) abnormal, (5) borderline, (6) diffuse convulsive disorder, (7) psychomotor, (8) no classification and (9) miscellaneous.

Missing teeth, excepting third molars, were recorded. Roentgenograms were not

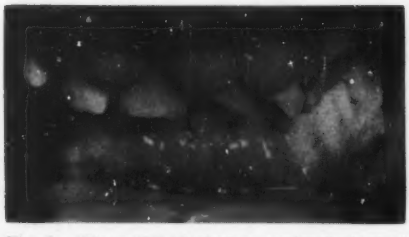

Fig. 8. (Stage III) Marked generalized gingival hyperplasia.

Fig. 9. (Stage IV) Very severe generalized hyperplasia.

employed. A note was made concerning the presence of appliances. The anterior maxillary and mandibular teeth were observed for fractures, and the teeth were numbered from left to right from cuspid to cuspid with numbers one to six. Fractured teeth were indicated by the number of the specific tooth in the space provided.

The amount of gingival hyperplasia is, of course, one of the main topics of this paper and the most time and effort was spent here. The mouth was divided, as illustrated in Figure 3, into six parts, each rated individually. The degree of hyperplasia was then recorded in four categories, as suggested by Harris and Ewalt:[19] (0) *normal* (Fig. 4) no reaction observed; (I) *minimal*, (Fig. 5) characterized by an increase in density of the gingivae, with marked stippling and a granular appearance; (II) *moderate* (Figs. 6 and 7) ("Quantitative changes consisted of an increase in the interdental papillae and a rolled effect of the gingival margin"); (III) *marked* (Fig. 8) ("Stage three was that of encroachment of the gingival tissues on the clinical crown of the tooth"); (IV) *very severe* (Fig. 9) ("Stage four is sufficient overgrowth of the tissues to interfere with function").

Oral hygiene ratings from 1 to 5 were recorded. The dental record was consulted to obtain the most recent date of prophylaxis.

The number of decayed, missing and filled teeth was recorded in the spaces provided. Additional abnormalities, such as excess body hair, also were noted.

The total of sums in columns 38 through 43 was recorded to give a *Hyperplasia Index* from 0 to 24 for each patient.

Part II of this article will be published in the January 1961 Journal of Periodontology.

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THIRD ANNUAL SUNSHINE SEMINAR

Greater Miami Chapter of Alpha Omega announces its Third Annual "Sunshine Seminar" at the Deauville Hotel, Miami Beach, Florida. Sessions begin January 20, 1961 and continue through the 23rd.

Clinicians for this year are Dr. Morton Amsterdam and Dr. Walter Cohen of the University of Pennsylvania, Dr. Harry Sicher of Loyola School of Dentistry, Chicago College of Dental Surgery and Dr. Vincent Trapozzano.

Theme of this year's seminar is "Occlusion." Tuition fee is $150.

All inquiries and reservations should be sent to Dr. Irving Gordon, 420 Lincoln Road, Miami Beach, Florida.

First Symposium on Oral Pigmentation*

CLIFTON O. DUMMETT, PROGRAM CHAIRMAN AND CONSULTING EDITOR

1. THE PHYSIOLOGY AND BIOCHEMISTRY OF PIGMENTATION
J. F. Volker and J. A. Kenney

2. GENERAL PIGMENTATION OF THE HUMAN ANATOMY
E. C. Sensenig

3. ORAL PIGMENTATION
C. O. Dummett

4. THE HISTOLOGY OF ORAL PIGMENTATION
T. E. Bolden

5. DIAGNOSIS OF PIGMENTATIONS OF THE ORAL TISSUES
E. Cheraskin

6. THE MELANOMATA
J. C. Ashhurst

7. AREAS OF RESEARCH IN ORAL PIGMENTATION
C. O. Dummett and C. T. Mason

INTRODUCTION

In the oral tissues, pigmentation is observed in a large number of individuals who evidence no pathosis upon examination. Also to be found are pigmentations of the oral tissues which result from various pathologic conditions, of both local and systemic etiology.

The relative dearth of scientific information on this interesting subject has stimulated conjecture and superstition about incidence, etiology, significance and relatedness. In dental textbooks the subject has been treated either minimally or not at all.

It is with the organization and evaluation of the accumulated material on pigmentation generally, and on the oral tissues specifically that this first symposium on oral pigmentation was especially concerned. The distinctive contributions to the sciences of oral diagnosis and oral medicine which would emanate from such material are obvious.

It has been possible to publish collectively the papers which were presented at this symposium. The amassed information has emphasized the need for new knowledge, and in addition, has indicated the direction towards which future research studies should be aimed.

CLIFTON O. DUMMETT, D.D.S.,
Chief, Dental Service, VA Hospital, Tuskegee, Alabama

Symposium Conducted at The Veterans Administration Hospital, Tuskegee, Alabama, November 20, 1959.

The Physiology and Biochemistry of Pigmentation

by JOSEPH F. VOLKER, D.D.S., PH.D.,* *Birmingham, Alabama* AND JOHN A. KENNEY, JR., M.D.,** *Cleveland, Ohio*

ALTHOUGH there has always been a great interest in the physiology and biochemistry of the skin pigmentation, the early workers in the field were greatly hampered by the lack of instruments suitable for measuring "in situ" normal and altered skin pigmentation. This difficulty was surmounted by the efforts of Edwards and Duntley[1] who were successful in adapting spectrophotometric techniques to analyses of the skin color. With their apparatus it became possible to record the reflectance of the intact living skin over the entire visible spectrum from the violet end at 400 millimicrons to the red end at 700 millimicrons. Typical findings of investigators[2] utilizing this form of equipment is seen in Figure 1. These observations should be of special interest to dental scientists because they are made on a facial structure, the cheek. They give encouragement to the possibility of making similar measurements on intraoral tissues.

Subsequently, Goldzieher, and his associates[3] were able to modify the basic instrument so that reflectance measurements could also be made in the near and far ultraviolet range with wave lengths of 220 to 400 millimicrons. Collectively, the researches to date indicate there are six primary pigments in the skin. They are (1) Melanin which has a marked absorption at the violet end of the visible spectrum but no clearly defined absorption band; (2) oxyhemoglobin which has definite absorption bands at 542 and 576 millimicrons; (3) reduced hemoglobin which has a single broad absorption band at 556 millimicrons; (4) carotene which has a well defined absorption band at 482 millimicrons; (5) melanoid which, like melanin, has general absorption at the violet end of the spectrum but also has a distinctive absorption band at 400 millimicrons; (6) soft keratin which has a characteristic absorption band at 290 millimicrons.

It is obvious that skin pigmentation is modified by physical conditions. If the dermis is thin, it is more transparent and permits the blood pigments to show through. In those instances where the dermis is thick its transparency is reduced and it tends to have a yellow appearance. It is quite probable that the Tyndall effect also influences skin color. This is a light scattering phenomenon that results from the rearrangement of light passing through a turbid medium. Light transmitted under these circumstances contains an increased number of the red long wave lengths since the light scattered to the side and back to the source has a greater per cent of the blue shorter wave lengths. Because the stratum corneum of the skin is relatively transparent, it is responsible for only minimal scattering of the light. The deeper layers of the skin which are more turbid, however, scatter light to a considerable degree. As a consequence, if a pigmented area is in the deeper layers of the skin, it appears bluer. This phenomenon is responsible for the so-called blue nevi.

Since melanin is the most important of the skin pigments, it has been the most intensively studied. It is present as coloration in many biologic forms including fishes, reptiles, and mammals. Some idea of its profound effect upon skin color can be gained from the knowledge that the total amount of melanin in an adult Negro man has been estimated to be only one gram. Chemically, melanin is a high molecular weight dye that is insoluble in water and most organic solvents. On analysis it yields approximately 57% carbon, 30% oxygen,

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Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama, on 20 November, 1959.

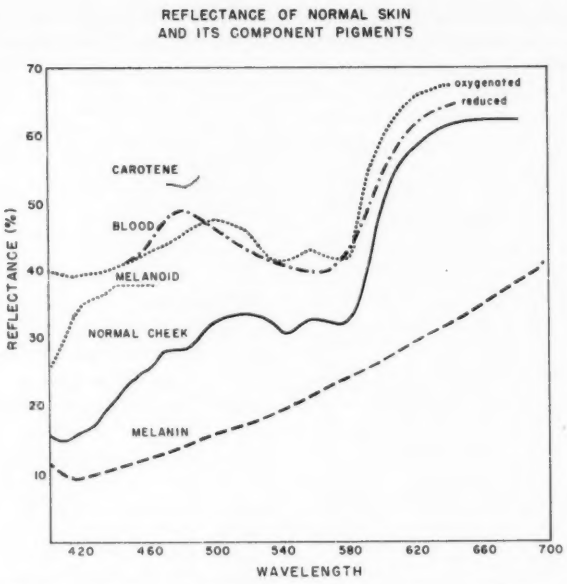

Fig. 1. Reflectance of normal skin and its component pigments.

9% nitrogen, and 4% hydrogen. It reduces silver and gold salts and is susceptible to oxidation by hydrogen peroxide and potassium permanganate.

Melanin is formed only in the cytoplasm of the melanin-forming cell, the melanocyte. These are dendritic or branched cells which are found at the epidermal-dermal junction of the skin and mucous membranes, in the leptomeninges of the central nervous system and in the uveal tract and retina of the eye. The overwhelming weight of evidence today supports the theory of the embryologic origin of melanocytes from the neural crest. Zimmerman and Cornbleet[4] studied the pigmentation of the Negro fetus and found melanocytes (by the Bodian reduction silver stain and the dopa reaction) in the third month of intrauterine life. The melanocytes were located in the intercellular epidermal spaces and formed an intricate pattern by their long processes. These cells are at first globular in shape, constituting melanoblasts, later becoming oval and dendritic, at which time they are called melanocytes.[5] These dermal melanocytes reach the epidermis and become epidermal melanocytes. Melanocytes remaining down in the dermis in the human being gradually become inactive. In the sacral region melanin is retained long enough for them to form the Mongolian spot. Blue nevi, already mentioned above, are formed by these dermal melanocytes. Rarely a melanoma may arise from such nevi.

The melanocyte is characterized by two or more dendritic processes and contains, in its pigmented form, numerous light or dark brown cytoplasmic granules. Recent electron microscopic studies of melanocytes in the human epidermis[6] have shown melanocytes to contain many more mitochondria and a more complex endoplasmic reticulum than the adjacent basal cells. The intercellular bridges, so characteristic of ordinary basal cells, are absent in melanocytes.

It is believed by Masson[7] that melanocytes are able to transfer melanin granules

into neighboring epidermal cells, a process which he terms "cytocrine" ability. This view is disputed by other authorities such as Montagna[8] who believe that as melanocytes migrate, melanin granules are picked up by the other cells of the epidermis. However, the cytocrine theory has found some support in recent electron microscopic investigations. Birbeck and his associates[9] found in studying the fine structure of hair melanocytes and cortical cells with the electron microscope that melanocytes had a type of cell polarity characteristic of a secretory cell. Saggital sections of the hair follicle at different levels revealed that a portion of the cytoplasmic process of the hair melanocyte appeared to be phagocytized by the cortical cell. Then the cell wall of the process disappeared and the melanin granules were seen to be dispersed throughout the cytoplasm of the cortical cells.

Melanocytes have a role in the protection of the skin against ultraviolet light. As epidermal cells move outward to become the stratum corneum, the melanin granules contained within them are carried along and appear in the stratum corneum not as granules any longer but as fine irregular pigmented particles. In dark-skinned individuals the stratum corneum is regularly flecked with these dust-like melanin particles. By contrast, the stratum corneum of Caucasians is not ordinarily melanized except during a short period after exposure to ionizing and ultraviolet radiation.[10] Thomson[11] has been able to show the protective function of melanin against ultraviolet light. Using some of the African hospital aides and white South Africans as subjects, he obtained layers of stratum corneum from unexposed skin of both sets of individuals by means of cantharides blisters. The Negro stratum corneum was distinctly the more melanized, even though the thickness of the stratum corneum in both the white and colored skin was of the same thickness. When these sheets of stratum corneum were placed on photographic plates and exposed to the ultraviolet light, the markedly greater filtering ability of the darker skin was clearly shown.

The work of Szabo[12] is outstanding with regard to the number and distribution of melanocytes. In general there are more melanocytes per square millimeter on exposed skin than on covered areas. However, there is a high density of melanocytes about the genitalia and the nasal mucous membranes.

Melanocytes are symmetrically distributed and do not differ significantly in size, shape and population density in various races. There are approximately two to three times as many melanocytes present in facial skin as elsewhere. The total epidermal melanocyte population is estimated at 2×10^9 with an average of 1560 per square millimeter $\pm$ 110 S.E.[10]

Variations in skin color between racial groups and possibly between different individuals of the same race are a function of the rate and amount of melanin production by melanocytes rather than of the number of melanocytes present. It has been pointed out that the cheek of a Caucasian with white skin color contains more melanocytes per square millimeter than the thigh of a dark-skinned individual with brown or black skin color.[10]

Around the turn of the century, Bourquelot and Bertrand demonstrated the presence of an enzyme in mushrooms that could oxidize the amino acid tyrosine to a black pigment. They named the enzyme tyrosinase. Shortly thereafter, vonFurth expressed the opinion that this oxidative enzyme was responsible for skin melanin formation. It was not, however, until 1950 that it was possible to demonstrate the presence of tyrosinase in the skin by histochemical methods.[13] The key material in melanin formation is 1-3, 4-dihydroxyphenylalanine which has the common name "dopa." This substance is the first compound formed by the action of tyrosinase and oxygen on tyrosine. The early fundamental studies of Bloch clearly showed that if frozen sections of pigmented skin

are incubated with "dopa" at pH 7.3 brown granules appear in the cytoplasm of scattered cells in the basal layer. The enzymatic nature of the reaction is easily established. It does not take place if the skin is boiled, if the pH is made acid or if an enzyme poison such as hydrogen sulfide is present. Whereas the conversion of tyrosine to dopa by oxygen and tyrosinase is relatively slow, the subsequent action of these materials on dopa initiates a series of rapid changes that result in ring closure and ultimately formation of Indole 5,6-quinone which furnishes the basic unit for melanin formation by polymerization. These complex changes[14,17] are illustrated in Figure 2.

Initially it was assumed that melanoid was a degradation product of melanin. This belief was supported by the observation that they were found usually in the same areas of the skin and that melanoid appeared after considerable quantities of melanin had been formed in response to prolonged exposure to sunlight. More recently, however, it has been shown that testosterone injections in castrates or eunuchs increase greatly the melanoid content of their skin with very little effect on melanin content. Also, melanoid formation of the skin of eunuchs is greatly stimulated by ultraviolet radiation without any unusual melanin production.

CAROTENE

Carotene contributes a deep yellow color to the normal skin. It will be recalled that this pigment bears a close relationship to vitamin A and is responsible for the yellow color of carrots and butter. The carotene content of the skin of women is usually higher than that of men. This is particularly the case in the breast, buttocks, and abdomen which are sites of minimal carotene deposition in males. This pigment is stored in the lipid-rich portions of the body, the dermal and subcutaneous fat, and the horny layer of the skin. Its presence in the latter structure is responsible for the yellow color of the soles and palms in clinical carotenemia. It has been reported that carotene is present in large quantities in the skin of untreated castrates and can be reduced by the administration of testosterone.

The role of the hemoglobins in skin coloration is obvious. Oxyhemoglobin contributes a strong red component where there is a rich arterial supply and the subpapillary-capillary plexus and the dermal papillae with their capillaries are close to the skin surface. Reduced hemoglobin is responsible for the bluish component of skin color, particularly in such areas as the dorsum of the feet and the lower parts of the trunk. It should be evident that the principal color patterns of the intraoral tissues are attributable to copious blood supply of the mouth mucous membranes.

It has been established that copper is necessary for melanin formation. The enzyme tyrosinase is in fact a copper-protein complex, with copper essential for the action of the enzyme. If potassium cyanide is added to tyrosinase and the copper cyanide complex is removed by dialysis, more than 80% of the enzyme activity is lost. It may be completely restored by adding cupric ions or partially restored by nickel ions. It has been demonstrated also that many copper binding agents effectively inhibit melanin formation. These include carbon monoxide, cyanide ion, hydrogen sulfide and a host of organic sulfur compounds, including such materials as cysteine, glutathione, thiourea and related compounds. There is evidence that these compounds have their inhibitory effect both "in vitro" and "in vivo."

Data has been accumulated that sunshine and x-rays stimulate skin pigmentation because they destroy the inhibitory sulfhydril groups of the skin. This in turn permits the tyrosinase to act freely on the enzyme substrates. A decrease in the sulfhydryl concentration in the skin as much as 83% has been observed following ultraviolet radiation of experimental animals. Utilizing histochemical techniques it

has been possible to show the presence of tyrosinase in the epidermis after ultraviolet irradiations have released it from its inhibited state.

Although a rather large number of chemicals have been shown to interfere with melanin formation by such obvious mechanisms as competitive inhibition, reaction with melanin precursors, and interference with the oxidation of melanin intermediates, the ability of some compounds of clinical significance to modify melanin formation remains obscure. Illustrative of this category is the monobenzyl ether of hydroquinone which is also known as Agerite alba. This compound is used in making rubber gloves and has been known to cause numerous cases of leukoderma. Investigation has shown that it requires approximately a month of skin contact for the depigmentation to become evident. Histologically there are no dopa positive cells in the areas of depigmentation. The oral administration of the material has been reported to have no effect on skin depigmentation. Practical use is made, however, of this depigmentation action of the compound by incorporating it in ointments and lotions for local applications to hyperpigmented areas of skin. Repigmentation takes several months following the removal of the agent. Similarly, little is also known of the manner in which many other materials and procedures such as nitrogen mustard and ionizing radiation cause local depigmentation.

Of greater interest to the practitioner are the effects of such biological materials as trace minerals, amino acids, vitamins, and hormones on pigmentation. The literature in this connection is very considerable and only a small segment of the investigations can be considered in this brief review.

Nutritional disturbances are known to influence pigmentation. It has been observed that amino acid deficient diets result in a loss of skin pigment. This would seem explainable on the basis of the amino acid needs of the tyrosine substrate and the enzyme protein.

Copper deficiency results in the loss of pigmentation, presumably because this ion is necessary for tyrosinase activity. This condition apparently may be reversed by pantothenic acid administration. The mechanism of this interrelationship is obscure but it is thought that pantothenic acid is needed to bring about the combination of copper with tyrosinase. This belief is supported by the finding that copper accumulates in the skin of pantothenic deficient animals. A deficiency of dietary zinc has been noted to cause depigmentation in experimental animals. This condition is reversed when trace quantities of zinc are added to the diet. Although large amounts of zinc are found in association with the melanin pigments of mammalian eyes, the possible interaction of copper, zinc and melanin is little understood. It has been reported also that the excessive ingestion of molybdenum causes depigmentation. This seems due to an interference of copper metabolism since an excess of dietary copper corrects the condition.

Although it is generally agreed that the ascorbic acid level of the skin influences pigmentation, the mechanism seems extraordinarily complex. It is known that ascorbic acid enhances the transformation of tyrosine to dopa by ultraviolet radiation. Yet ascorbic acid has been observed to prevent further oxidation of dopa to dopa quinone by its reducing action. Ascorbic acid has been shown to be capable of reducing dark oxidized melanin to its lighter reduced form and it has been suspected of the limiting of pigmentation in Addison's disease. There is evidence that local injection in high concentration prevents sun tanning. Data also have been accumulated that indicate ascorbic acid is a major factor in maintaining the sulfhydryl level of the blood and tissues. It is hoped that future researches will clarify these relationships.

During the last twenty-five years many investigations have been concerned with the relationship of the vitamin B complex

ENZYMIC OXIDATION OF TYROSINE TO MELANIN

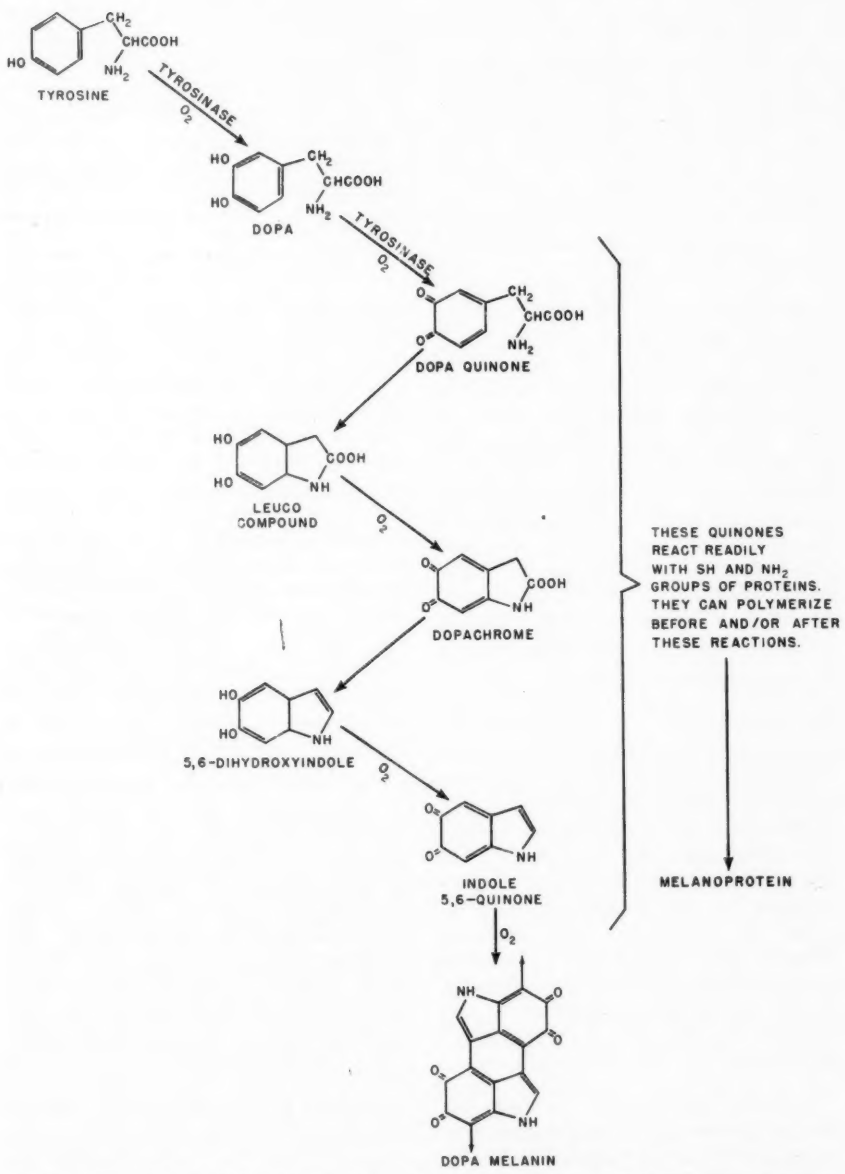

Fig. 2. From—Lerner, A. B. and Case, J. D.: Pigment Cell Regulatory Factors. J. Invest. Dermat., 32:211, 1959.

to pigmentation. Initially it was observed that a deficiency of the filtrate factors of the vitamin B complex resulted in a depigmentation in experimental animals that could be reversed. Although pantothenic acid seemed to be the principal substance involved, there was indication that other deficiency factors were of importance. Later it was shown that folic acid and biotin were also part of the deficiency syndrome. In the absence of these latter substances apparently either the intestinal flora cannot synthesize pantothenic acid or the tissues cannot utilize it.

Numerous studies have been directed at an understanding of the relationship between hormonal factors and melanin formation. Indeed, most of the emphasis in recent investigations has been placed on this aspect of the subject. Hormones from the pituitary and pineal glands have been found to have an important bearing on pigmentation. Alpha and Beta melanocyte stimulating hormones (MSH) have been isolated from hog pituitary glands.[15] These substances have been found to be polypeptides that are smaller than, but related in structure to hog ACTH. They exert a powerful darkening effect on frog skin and have been found to darken human skin in vivo and in vitro. Lerner and his associates have recently isolated from the pineal gland of cows a powerful lightening agent, named melatonin.[16] This is a 5-hydroxyindole compound which is probably derived from the metabolism of the amino acid tryptophan. These workers have been able to demonstrate melatonin in human pineal glands and in bovine hypothalamus and peripheral nerves. Although melatonin is the most powerful lightening agent known acting on frog skin, its role in human pigmentation is not yet clarified.[17]

In lower vertebrates it has been repeatedly shown that melanin formation is influenced by thyroid hormone. Indicative of this reaction is the failure of tadpoles to develop pigment when the hormone is absent. It has been observed also that hyperthyroidism in man is often accompanied by pigmentation. Since it is known that thyroxin generally stimulates oxidation, this phenomenon may be explained in part by the acceleration of some of the steps involved in the conversion of tyrosine to melanin. It is of particular interest to note the similarities between thyroxin and melanin. Both utilize tyrosine as a substrate, both are inhibited by copper binding agents and both are subject to regulation by pituitary tropic hormones.

There is substantial evidence that the adrenal hormones are regulators of melanin formation. Addison's disease, a condition which particularly affects the adrenal cortex, is accompanied by a characteristic melanosis. This melanosis develops slowly and is particularly marked in the warm areas of the skin such as the axilla, the pressure points, and the exposed part of the face and extremities. This suggests that increased skin temperature may also be involved in melanin formation. In experimental animals which show deep pigmentation because of filtrate factor deficiency, adrenalectomy causes repigmentation. This effect can be counteracted or inhibited by administering desoxycorticosterone. Contrary to the expectation of many researches, ascorbic acid therapy is unable to reverse Addisonian melanosis.

The gonadal hormones stimulate melanin pigmentation. Eunuchs usually have pale skin and are resistant to tanning. When they are given testosterone and subsequently exposed to the sun, however, strong skin pigmentation occurs. It has also been observed that the unilateral application of estrogens to sites such as the nipple results in unilateral tanning. It is generally believed that gonadal hormones exert their action by stimulating the melanocytes to form melanin.

It should be reiterated that only selected examples are being included in this presentation and that these merely illustrate the extraordinary complexity of the factors that influence skin melanin content. Some idea of the scope of the problem can be

TABLE I
Classification of Melanin Pigmentation of the Skin[18]

Melanosis Not Associated with an Increased Number of Melanoblasts			*Melanosis Associated with Increased Number of Melanoblasts*
External Causes	*Internal Causes*		
	Conditions Primarily of General Medical Interest	*Conditions Primarily of Dermatologic Interest*	
Sunlight Ultraviolet rays Roentgen rays Alpha rays from thorium X Photosensitization to actinic rays Heat rays Mechanical irritation Chemical irritation Vagabonds' disease	Vitamin A deficiency* Vitamin C deficiency* Pellagra* Sprue* Anemias* Lymphomas* Gaucher's disease* Niemann-Pick's disease* Acanthosis nigricans Scleroderma Ingestion of arsenic Vagabonds' disease*† Ochronosis (alcaptonuria)*‡ Addison's disease Acromegaly* Cushing's disease* Hyperthyroidism* Pregnancy Chloasma uterinum Hemochromatosis (many cases)* Additional miscellaneous causes*	Erythrose peribuccale pigmentaire Postinflammatory condition Chronic dermatitis Dermatitis herpetiformis Urticaria with pigmentation Urticaria pigmentosa Xeroderma pigmentosum War melanosis (Riehl) Lentigo Additional miscellaneous causes	Ephelides (freckles) Pigmented nevus Neoplasms Melanoma Melanotic epithelioma Melanotic carcinoma Blue nevus Neurofibromatosis Albright's syndrome* Leschke's syndrome* Congenital neurocutaneous syndrome of melanosis of skin and central nervous system* Skin pigment spot localized over spina bifida occulta* Mongolian spot

* Diseases added to original classification, on the basis either of data in the literature or of their similarity to disorders already classified.

† Vagabonds' disease may have an internal as well as external cause.

‡ Pigmentation chiefly in cartilage; skin pigmentation less constant and in corium originates from homogentisic acid.

gained from the outline information compiled by Jeghers as seen in Table 1.[18]

In any discussion of pigmentation it is impossible to avoid comment on sun tanning. This phenomenon involves at least three recognizable effects. The first of these is an initial darkening which appears a few minutes after exposure and reaches a maximum in an hour. It is believed to be caused by the long wave ultraviolet spectrum between 300 and 420 millimicrons and represents a photo-oxidation of the preformed reduced melanin. This does not occur if the skin is blanched by pressure, presumably because this procedure diminishes the oxygen supply to the tissues. The second effect is noticeable several days after exposure to the sun and is characterized by a surface migration of the melanin granules in the epidermis. In certain instances this is so pronounced that the basal layer becomes relatively free of melanin granules. Ultimately a third stage, the formation of new melanin, may be recognized in sun tanning. This reaches a maximum after two and a half weeks and begins to decline after one month and returns to the normal melanin level nine months later. There is evidence that the new melanin formation is the result of the action of the shorter ultraviolet radiation of 280 to 310 millimicrons. Radiations of this nature have the characteristic of producing erythema.

Some of the so-called sun tanning agents, such as paraminobenzoic acid, have the ability to filter out the erythema-producing radiations but allow the pigment-darkening rays of greater than 320 milli-

microns to pass. Others, such as 8-methoxypsoralen, increase the pigmentogenic effect of sunlight by sensitizing to ultraviolet radiations of wave lengths above 320 millimicrons. This effect is taken advantage of in therapeutic use of the psoralens in treating vitiligo.

Very little attention has been paid to pigmentation of the oral structures. A notable exception has been the researches of Dummett[19] which have been concerned primarily with the pigmentation of the oral mucous membranes. It is entirely probable, however, that studies directed at other aspects of oral pigmentation might yield valuable data. It is known, for example, that mottling of the enamel resulting from the excessive ingestion of fluorides at the time of calcification is initially characterized by zones of depigmentation. Subsequently, these areas become pigmented and proceed through a series of color changes from light yellow to a reddish brown and then to black. These changes are particularly pronounced on those surfaces of the teeth that are exposed to light, such as the labial surfaces of the upper anterior teeth. A possible explanation of this phenomenon would be that salivary or food amino acids permeate the defective enamel surface and are acted upon subsequently by tyrosinase-like enzymes of bacterial or salivary origin in the presence of ultraviolet radiation. This results in progressive melanin formation. It is of interest that these pigments, although superficial, are resistant to the action of almost all solvents but can be removed by the action of concentrated solutions of hydrogen peroxide.

Similarly, it is known that the carious process, particularly in the dentin, is associated with pigment formation. These pigments are in the yellow, brown, black categories. Deakins[20] has analyzed these materials and has presented data which indicate that they might very well be classified as melanins. Dreizen and his associates[21] have extended these observations and have accumulated evidence that the pigmentation is a melanoidin. According to them, the latter material is formed by the interaction of bacterial degeneration products of glucose and tooth proteins.

Since it is well known that the arrest of caries of the enamel and dentin is often associated with increased pigmentation, the attention of investigators might be directed profitably to a study of yet another form of oral pigmentation.

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General Pigmentation of the Human Anatomy. A Summary

by E. CARL SENSENIG, PH.D., *Birmingham, Alabama*

1. Classification of pigments. Exogenous pigments were excluded as the emphasis was placed on pigments formed within the human body.

Endogenous pigments were discussed under hematin, hematoidin, lipochromes, hemoglobins and melanins. Each of these was briefly mentioned with the melanins discussed in more detail. The melanins while being the characteristic pigment of the human skin are also present in the pia mater, choroid cells of the eye, iris epithelium, substantia nigra of the mid-brain, pineal and pituitary glands and in the medullary cells of the adrenal gland.

2. The skin was illustrated with lantern slides and its structure was discussed under stratum germinativum which is further subdivided into stratum basalis and spinosum. It is within this layer and more specifically in the deeper division (stratum basalis) where the pigmented dendritic cells are located. The dendritic cells, the producers of the melanin pigment, can be best demonstrated by the Dopa reaction. In deeply pigmented individuals the pigment extends throughout the stratum germinativum and even into the stratum granulosum. The superficial strata lucidum and corneum were briefly mentioned.

3. The embryology of the human skin and the appearance of pigmentation within the skin was discussed. It was pointed out that the present knowledge of the embryology of the human skin and its contained structures was inadequate. On the basis of experimental evidence, however, the melanin forming cells, dendritic cells, can be considered as of neural crest origin.

4. Physical Anthropology. The use of skin pigmentation in classifying the races of man was discussed. Within the three major races of man that is Black or Negroid, Yellow or Mongoloid and White or Caucasoid, the Negroid and Mongoloid show less degree of color variation than the Caucasoid. The White race shows all extremes from the very fair Scandinavian to the very dark Caucasoid ethnic groups of north and east Africa.

Department of Anatomy, University of Alabama Medical Center.

Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama, on 20 November, 1959.

Oral Pigmentation

by CLIFTON O. DUMMETT, D.D.S., *Tuskegee, Alabama*

THE physiology, biochemistry and anatomy of general pigmentation have been adequately considered, so that the singular facet of oral pigmentation can now be introduced.

A differentiation should be made between pigmentations and color changes affecting the oral hard tissues, and those which affect the oral soft tissues. Insofar as the former are concerned, it should be noted that the physical character of the enamel surfaces of the teeth make it impossible for deep external straining to occur. It has been demonstrated that it is the superficial organic deposits which become discolored and show up as stains.

EXTRA AND INTRAORAL PIGMENTATION

In the oral soft tissues, melanin pigmentation is observed in a large number of individuals who show no pathosis upon careful examination. For the sake of convenience, these oral tissues are divided anatomically into intraoral and extraoral parts.

The lips comprise the extraoral tissues, and are inclined to exemplify rather spectacular variations both in form and appearance (Fig. 1). The transitional zone between the skin covering the outer surface of the lip and the true mucous membrane lining the inner surface, is the red area or vermilion border. The transitional region is characterized by numerous, densely arranged, long papillae of the lamina propria.[1] Eleidin in the epithelial cells makes them translucent. The red color of the lips is due to the blood which is visible through the thin parts of the transparent epithelium. The extraoral tissues may be and often are affected with melanin pigmentation in the bases of the interdental papillae.

Comprising the intraoral tissues are the gingivae and gums, the cheeks, hard palate, tongue, soft palate and floor of the mouth. Melanin pigment may be found clinically in either one or all of these intraoral tissues. When present, it is found with greatest intensity in the gingivae (Fig. 2). Next in order of decreasing intensity are the cheeks (Fig. 3), the hard palate (Fig. 4), the tongue (Fig. 5), the soft palate (Fig. 6) and finally the floor of the mouth (Fig. 7).

Fig. 1. Labial tissues showing melanin pigmented and non-pigmented areas of the lower lip.

NORMAL GINGIVAL COLOR

The gingivae are the most frequently pigmented of the intraoral tissues in addition to being the most readily seen during inspections. Because of these factors it is likely that they would play an important part in the diagnosis of oral disease. The accuracy of description of the normal gingivae is, therefore, a most important prerequisite to the diagnosis of disease.

The variations that occur in the color of the gingivae of some Caucasians and so many non-white races would suggest that the frequent descriptions of the normal gingivae as 'coral pink' are subject to question, and from a pandemic standpoint, these descriptions are incorrect.

Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama on November 20, 1959.

Fig. 2. Moderately intense melanin pigmentation of the gingivae.

Fig. 3. Melanin pigmentation of the buccal mucosa.

Contrary to general opinion, oral pigmentation, and specifically gingival pigmentation, is by no means confined to members of the Negro race. It has been reported in the French, Filipino, Algerian, Arabian, Chinese, East Indian, German, Roumanian, Italian, Jewish, Greek, Puerto Rican, Syrian, Peruvian and Ceylonese.[2] Moreover, clinically non-pigmented oral tissues are frequently found in members of the Negro and other heavily pigmented races (Fig. 8).

Physiologic oral pigmentation is therefore not limited to any one race, although it is a common characteristic of the more heavily pigmented races. Becker[3] demonstrated microscopically the presence of epidermal pigmentation in the normal buccal mucosa of 74% of 53 unselected autopsies. Clinically, pigmentation was absent. Becker's cases were all Caucasians and most of them were of medium complexion. He concluded that pigmentation of the buccal mucosa was a normal finding.

A detailed consideration of this particular aspect of oral pigmentation would suggest the following definition as a more accurate statement of the patterns of normal gingival pigmentation:

"The color of the healthy gingivae is variable, ranging from a pale pink to a deep bluish purple. Between these limits of normalcy are a large number of colors which depend primarily upon the intensity of melanogenesis, the degree of epithelial cornification, the depth of epithelization and the arrangement of gingival vascularity. Moreover, color variations may be uniform, unilateral, (Fig. 9) bilateral, mottled, macular or blotched, and may involve the gingival papillae alone or extend throughout the gingivae and into other oral tissues. Nonpigmented gingivae are found more often in fair-skinned individuals, while pigmented gingivae are usually seen in dark-skinned persons."[4]

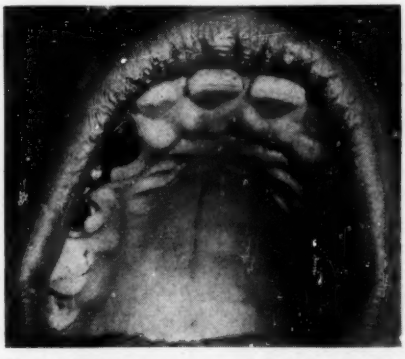

Fig. 4. Melanogenous central line in the hard palate.

Fig. 5. Scattered pigmented maculae of the dorsum of the tongue.

Fig. 7. Pigmentation of the floor of the mouth.

ABNORMAL ORAL COLOR CHANGES

Abnormal color changes do affect the oral hard tissues. The most common surface stains result from the use of tobacco, which influences the organic deposits on the enamel and not the enamel itself. Similar superficial pigmentary changes may result from medicines containing iron or other metallic preparations, foods, stimulants and condiments. Green and black pigmentary changes around the necks of children's teeth have been described[5] and have been said to be due to bacterial growths. The deep penetration of pigments into the hard structures occurs when the enamel has been affected by such conditions as dental caries, hypoplasia, dental fluorosis or circumrestorational seepage.

Intrinsic pigmentations of the dental calcified tissues may occur as a result of the penetration of pigments from the pulp tissues of the teeth (Fig. 10). There are changes in tooth color which result from tooth devitalizations resulting from trauma, endocrine disturbances, the blood dyscrasias, or from diseases of the liver.

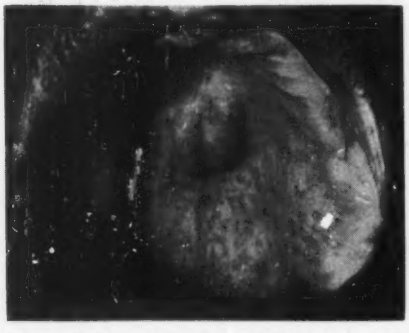

Fig. 6. Diffuse pigmented maculae of the soft palate.

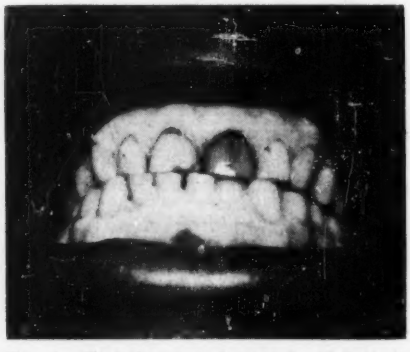

Fig. 8. Clinically non-pigmented gingivae in Negro patient with intense cutaneous pigment.

Fig. 9. Unilateral mottled gingival pigmentation.

Fig. 11. Gingival color changes from irritating effects of calculus.

There are also color changes which are due to systemic conditions occurring during periods of tooth development in which all the teeth may be equally affected.

Age too has been listed as one of the factors involved in oral hard tissue color changes, and there has been described[6] a tendency of teeth to "yellow with age."

Insofar as abnormal color changes in the oral soft tissues are concerned, it should be pointed out that pigmentation may occur in these tissues as a result of disease.[7] From the local standpoint, color changes in the gingivae may result from the inflammatory effects of irritants (Fig. 11). These effects are the complex, vascular, lymphatic and local tissue reactions produced by various irritants. In addition, there are the more complicated mechanisms by which pigmentation arises from systemic disturbances. These pigmentations are not localized. They are intrinsic in character as opposed to the extrinsic nature of the color changes resulting from irritants.

Although probably not as obvious as in

Fig. 10. Penetration of stains and pigments into tooth structure.

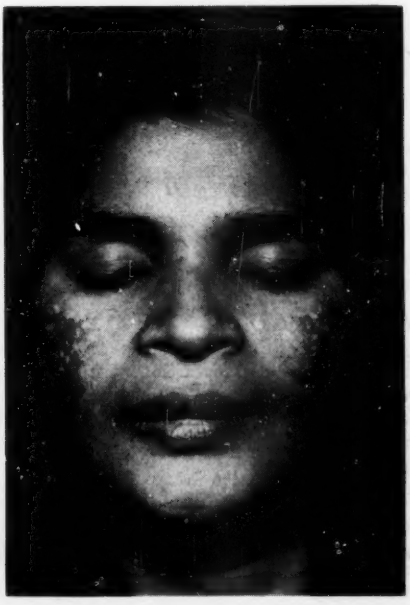

Fig. 12. Pigmentary changes associated with chloasma.

the gingivae, abnormal color changes frequently affect the remaining tissues, especially the tongue. The coated tongue is often described as being white or light yellow in appearance, and a brown or black tongue is not a rare occurrence. In the blood dyscrasias, metallic poisonings, Addison's disease and Peutz-Jeghers syndrome, the remaining oral tissues may be and often are affected by pigmentary changes.

Extraorally, abnormal color changes also occur. Figure 12 shows pigmentary changes on both cheeks of a 23 year old Indian female. The patient insisted that these melanotic spots first appeared only 8 months prior to her being seen by the author. A medical diagnosis was made of chloasma, a condition in which a patchy melanosis occurs usually on the face, but occasionally elsewhere on the body. Often first seen during pregnancy, the condition is also seen in association with malaria, sprue and tuberculosis. This patient was not affected with any of these conditions at the time of examination. Investigators[8] have reported a predisposition to a chloasma-like melanosis in Hindus and African Negro females. The condition is persistent and is aggravated by exposure to sunlight.

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Book Review . . . .

Periodontal Therapy, by Henry M. Goldman; Saul Schluger; Lewis Fox; and D. Walter Cohen, Second Edition, 658 pages with 225 illustrations and 117 plates, index, $18.75. C. V. Mosby Company, St. Louis, Missouri, 1960.

The second edition of *Periodontal Therapy* continues as an excellent reference text for the student, general practitioner, and the specialist in the field of periodontia. The book is profusely illustrated with photographs and plates showing the many procedures now accepted for periodontal treatment. Indications, contraindications and limitations of the various methods of treatment are carefully explained.

Additions have been incorporated into the seemingly excellent first edition. Since diagnosis and treatment planning is such an important phase of periodontal therapy, the authors enlarged this chapter. Also, the portion devoted to surgery was developed to a greater extent, particularly that dealing with vesticular alteration.

The second edition of *Periodontal Therapy* is highly recommended for those who are interested in the development of skill in the treatment of periodontal involvements.—*H.M.S.*

Histology of Oral Pigmentation

by THEODORE E. BOLDEN, D.D.S., PH.D.,* *Jersey City, New Jersey*

THE histology of oral pigmentation probably does not differ generically, functionally or anatomically[1] from pigmentation elsewhere in the body. However oral pigmentation becomes an important clinical consideration when it is remembered that the first signs of a systemic disturbance, and of a local disturbance too, frequently present in the oral cavity, and as changes in pigmentation.[2]

Pigmentary changes may be observed in both the hard and soft tissues of the oral cavity. This statement is concerned only with the soft tissues. The specific subject, histology of oral pigmentation can be understood best, perhaps, if two terms are defined: first, pigmentation and the factors which contribute to it, and second, the soft tissues of the oral cavity, or the oral mucosa, its distribution, function and structure.

PIGMENTATION, AND THE FACTORS WHICH CONTRIBUTE TO IT

Variations in skin color have been shown to depend upon the relative thickness of the skin, the light scattering phenomenon, and upon the amount and distribution of melanin in the skin,[3,4] the vascular bed, and its inherent color, yellow.

Primary Pigments. According to Lorincz ('54) the color of the normal human skin takes its origin from five primary pigments[5] (Table 1). Each pigment imparts a specific component, and by reflectance techniques has been shown to possess a different absorption band. These original spectrophotometric observations of Edwards and Duntley[6] have been confirmed by other workers using similar, but more refined techniques.[3]

Melanoblasts and Melanophores. Melanocytes (melanoblasts) are the normal mammalian melanin forming cells. They are of neuro-ectodermal origin, appear first in the dermis and then migrate to the basal layer, and produce melanin from tyrosine through the action of tyrosinase.[7] In human mucosa and skin,[5] they are found in the basal layer of epithelium as "clear cells" and as dendritic cells.[8] They have no intercellular bridges.[9] Dendritic cells form a syncytium around contiguous cells. Some melanocytes have been shown to move up gradually into the prickle cell layer, granular layer and finally horn layer, losing almost all of their dendrites but retaining much of their enzymatic activity.[8] Primitive (stem) cells are called melanoblasts.

Melanin containing cells in the lamina propria generally do not produce the pigment they carry and are known as melanophages, melanophores or chromatophores.

Melanin. The structure of melanin granules from pigment cells of human skin have been shown to have uniform size and shape,[5,6] within a given locus. They are round and approximately 0.3 microns in diameter, with a molecular weight of 20,000, are combined with a protein, a pseudoglobulin[7] and in vivo are insoluble in water and organic solvents, and are soluble in hot aqueous sodium hydroxide.[11]

Melanin granules have been shown to vary according to the size of the dendritic cell which carries them. Dendritic cells of the mucous membrane of the mouth are larger than dendritic cells from the epidermis of the nipple, axilla and abdomen and contain larger melanin granules. The granules are seen within the processes as well as within the cell body.[12]

Whether melanin granules in humans represent mitochondrial fractions or mitochondria is not quite clear. Transplantation

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Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama, on 20 November, 1959.

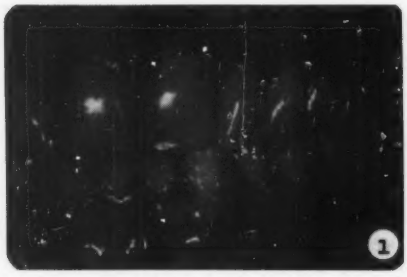

Fig. 1. Intraoral photograph showing pink masticatory (gingiva) and lining (alveolar) oral mucosa. Normal.

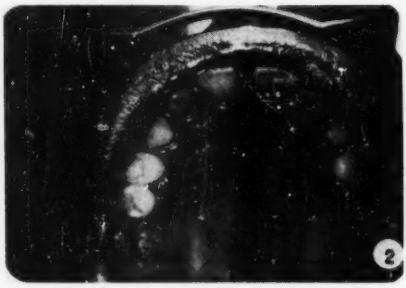

Fig. 2. Intraoral photograph showing diffuse pigmentation of the palatal and gingival mucosa. Normal.

experiments of the skin would tend to discredit this concept.[13]

SOFT TISSUES OF THE ORAL CAVITY—THE ORAL MUCOSA

Distribution. The oral cavity is lined by a mucous membrane which is continuous on its inner surface with the mucous membrane which lines the pharynx and on the outer surface with the skin. It consists of an outer stratified squamous epithelium and an inner lamina propria. Minor salivary glands are distributed throughout the mucous membrane.[14] The tissues are very vascular and have a rich lymph supply.

Function. The oral mucosa has been divided into three different types, primarily on the basis of function.[15] These include (1) the masticatory mucosa, which covers the hard palate and the gingiva, (2) the specialized mucosa, which covers the dorsal surface of the tongue and (3) the lining mucosa, which covers the soft palate, cheeks, vestibular fornix, upper and lower alveolar process peripheral to the gingiva proper, the inferior surface of the tongue, the lips and the floor of the mouth up to the inner surface of the lower alveolar process.

Structure. Certain structural differences exist between the various components of the oral mucosa, and between the oral mucous membrane and the skin (Table 2). The primary differences, however, are in the thickness and number of layers of epithelium, the absence, or presence and location of various epithelial derivatives, and the relative thickness of supporting connective tissue, its vascularity and mode of attachment.

Histologically the epithelium of the oral mucosa consists of four layers,[2] a stratum germinativum or basal layer, a stratum spinosum or prickle cell layer in which well defined intracellular bridges are visible, and which contains some mitotic figures, a stratum granulosum, and a

TABLE 1.
Skin Pigments and Their Absorption Bands.*

Melanin—brown or black.	Violet end of visible spectrum. No clear cut absorption band.
Oxyhemoglobin—red.	Sharp absorption bands at 542 and 576 millimicrons.
Reduced hemoglobin—dark blue or purple.	Single broad absorption band at 556 millimicrons.
Carotene—yellow. Areas rich in lipid.	Distinct absorption band at 482 millimicrons.
Melanoid—brown or black. Cornified areas.	General absorption at violet end of spectrum; distinct absorption band at 400 millimicrons.

*Compiled from a report by Edwards and Duntley.

TABLE 2.

Comparison of Histologic Features of Skin and the Oral Mucosa.

Oral Mucosa	*Area Covered*	*Covering Epithelium*	*Lamina Propria*	*Submucosa*
Lining Mucosa	Soft Palate	Non-keratinized, stratified squamous.	Papillae are short. Highly vascular with distinct layer of elastic fibers.	Relatively loose. Contains mucous glands.
	Inner surface of tongue.	Non-keratinized, stratified squamous.	Papillae are short.	Distinct separate layer not seen. Bound to connective tissue which invests muscle of tongue.
	Floor of mouth.	Non-keratinized, stratified squamous.	Papillae are short.	Loosely attached. Contains adipose tissue and sublingual glands (Mucous and serous alveoli).
	Lip	Non-keratinized, stratified squamous.	Dense connective tissue. Papillae are irregular.	Dense collagenous bundles with adipose tissue and mixed salivary glands. Firmly attached orbicularis oris.
	Cheek (buccal and labial mucosa).		Dense connective tissue. Papillae are irregular.	Dense collagenous bundles with adipose tissue. May contain sebaceous glands. Firmly attached to buccinator muscle. Glands between muscle bundles.
	Vestibular fornix. Alveolar mucosa.	Non-keratinized, stratified squamous. Thin, non-keratinized Ridges low and may be absent.	Dense connective tissue. Papillae are irregular. Papillae are low. Numerous vessels close to the surface. Many thin elastic fibers.	Loose connective tissue with adipose tissue. Well defined layer of loose connective tissue. Numerous thick elastic fibers. May contain mixed glands. Loosely attached to periosteum.
Masticatory Mucosa	Gingiva.	Stippled, thick, keratinized with granular layer. May be non-keratinized, or parakeratotic. Ridges are high. Basal cells may contain pigment granules especially bases of interdental papilla. Stratified squamous.	Papillae are high, slender and numerous. Dense connective tissue with low vascularity, few elastic fibers. Firmly attached to periosteum and to cementum.	No glands. No submucosa. Firmly attached to bone.
	Hard Palate.	Thick, keratinized, stratified squamous. Long papilla.	Dense connective tissue. Fixed to periosteum.	Variable. Same as gingiva at margins and in median zone. Adipose tissue and glands in irregular spaces of anterior and posterior regions.
Specialized Mucosa	Dorsal surface of tongue.	Keratinized, with various papillae some of which contain taste buds projecting from surface.	High papillae which support the surface projections. Von Ebner's mucous glands.	No definitive layer. Bound to connective tissue which invests muscles of tongues.
Skin	Entire Body.	*Epidermis*. Variable keratinized, stratified squamous. No blood vessels.	*Dermis*. Dense connective tissue. Contains epidermal appendages; sweat glands, hair follicles, sebaceous glands, and nails. Rests on hypodermis.	*Hypodermis*. Subcutaneous tissue upon which skin (Epidermis and dermis), rests and is attached by irregularly spaced bundles of collagenic fibers.

TABLE 3.
Histology of Oral Pigmentation—Normal

Author Date	*Condition Source Age, Sex*	*Area of Oral Cavity Involved*	*Clinical Status*	*Histology*
Adachi 1903	White	Buccal mucosa	Non pigmented	Pigment present in some cases.
Bonnet, Lebeuf 1921	French 60, M	Cheek, lips	Numerous black spots	Not given.
Ramel 1925	French	Oral mucosa	Non pigmented–5 Pigmented–7	Dopa positive–2 Dopa positive–6
Becker 1927	Autopsies-53	Buccal mucosa inner surface left cheek	Various degrees of pigmentation	Pigmentation present in epithelium and chromatophores.
Laidlaw, Cahn 1932	Negroes & Caucasians	Gum	Non pigmented areas	Melanin or melanoblasts or both were found. Melanoblasts were branched or dendritic.
Monash 1932	Negro	Gums, tongue, cheeks, hard palate	95%-areas of pigmentation 5%-non pigmented	Not given.
Breijer, Lignac 1952	Dutch 1-Colored 51-White	Gum	Non pigmented	Histologic and chemical demonstration of melanin in melanoblasts, in spotted form, in diffuse form in basal cell layer.
Orban, Wentz 1955		Gingiva, palate	Dark spots, streaks	Clear cells, located in basal layer, DOPA negative. Melanoblasts, dendritic cells, produce melanin, DOPA positive. Melanophores, macrophages which have picked up melanin, DOPA positive.

stratum corneum, where dead or dying cells accumulate, produce either a parakeratotic or keratotic layer, and finally are lost. The lamina propria consists of loose or dense collagenous fibers with deep or short papilla, varying degrees of vascularity, nerves and tissue cells.[16]

ORAL PIGMENTATION

Clinical Appearance. Normally the oral mucosa is pigmented,[19-22] red,[15] or pink[17,18] of smooth consistency, moist and very sensitive. Structural differences probably cause the difference in color between the pale pink masticatory mucosa of the gingiva, and the red lining mucosa, which shows numerous vessels close to the surface.[15] A greyish tinge to the gingiva has been attributed partly to differences in the thickness of the stratum corneum,[15] but may also be attributed to stagnation and poor oxygenation of oral tissues. (Figures 1, 2, 3).

Spectrophotometry. Spectrophotometric analysis of the oral mucosa probably would yield the presence of the same primary pigment components as found in the skin (Table 1) though perhaps in different proportions. Melanin may play the larger role even as it does in skin pigmentation.

Identification of Melanin. Sections of oral mucosa stained with hematoxylin and eosin may show the distribution of both

Fig. 3. Intraoral photograph showing variations in normal pigmentation of the oral mucosa. Maxilla—the masticatory mucosa is deeply pigmented, while the lining mucosa is pink. Mandible—there is patchy pigmentation of the masticatory mucosa.

Fig. 4. Photomicrograph from section of oral mucosa clinically pigmented. Note the "clear cells," melanoblasts and chromatophores. Hematoxylin and eosin. Magnification X190.

"clear cells" and dendritic cells within the basal layer of the epithelium (Figures 4, 5), and of chromatophores within the papilla of the lamina propria. A few granules may be seen within the cytoplasm of some clear cells (Figure 6) and fine yellow granules or golden granules appear to be distributed around the epithelial cells like a cuff. These granules are actually within the cytoplasm of dendritic cells and in many instances obscure the nucleus.

The same distributional pattern of melanin is seen in thionin treated sections. The granules stain black or yellow. An occasional dendritic cell may be seen to advantage in these preparations (Figures 7, 8, 9). Silver impregnation (Masson-Fontana) identifies melanin as a black precipitate which stands out sharply against the red color of the connective tissue (Figure 10). Melanin becomes positive with DOPA due to the oxidation of tyrosine. Tyrosinase activity is demonstrated by the black staining of melanin granules.

Melanoblasts, Melanin and Mast Cells. The mast cell is present in clinically normal and pathologic gingival tissue and may lead to some confusion in identification in some histologic preparations. Mast cells are found principally in the corium, but also constantly in the epithelium.[23] These cells contain PAS positive granules, exhibit strong pleomorphism, and in some instances show direct nuclear division.

With thionin, the granules of mast cells stain metachromatically, while melanin granules stain orthochromatically. A new technique recently developed selectively

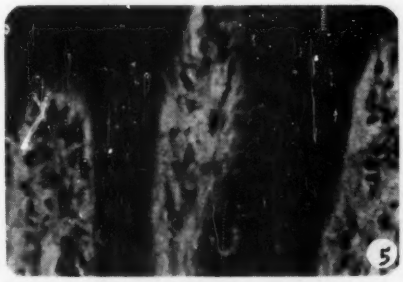

Fig. 5. Higher magnification through area shown in Fig. 4. Numerous chromatophores and one "clear cell" are seen to advantage. Hematoxylin and eosin. Magnification X850.

Fig. 6. Photomicrograph through epithelial ridge and connective tissue papilla. Numerous fine yellow-brown granules surround the basal cells like a syncytium. A few granules appear to be in the cytoplasm of the "clear cell." Hematoxylin and eosin. Magnification X1500.

TABLE 4.

Histology of Oral Pigmentation—Hyper- and Hypo-Pigmentation

Author Date	*Condition Source Age, Sex*	*Area of Oral Cavity Involved*	*Clinical Status*	*Histology*
Hudelo, Rabut 1927	Suprarenal dysfunction 24, M	Mucosa of cheek, dorsum of tongue, lips, gingiva	Round pigmentation	Cellular infiltration by a pigment with histo-chemical characteristics of melanin.
Oard, Kaletsky, Gettinger 1939	Addison's disease	Gum, maxilla floor of mouth	Pale brown to black spots or streaks	Brown refractile granules in stratum germinativum and in occasional histiocyte and polyblast. No increase in dopa oxydase activity.
Orban, Wentz 1955	Addison's disease	Oral mucosa, lips tongue, entire palate, gingiva cheeks	Pale brown to black spots or streaks	Hyperpigmentation with melanin deposited in the cytoplasm around nuclei of basal cells, and in macrophages. No increase in dendritic cells.
Ramel 1925	Melanoderma (2 cases) Idiopathic pigment (2 cases)	Oral mucosa		DOPA positive. DOPA positive.
Milian, Lenormand 1927	Syphilis	Buccal mucosa, gingiva	Round, melanotic well limited spots in juxta-position to leukoplakic lesions	Not given.
Milian 1928	Syphilis (?) F's only	Peribuccal dermatosis	Large black or brown spots on chin and internal part of lower lip perinasally	Few pigmented cells, only chromatophores.
Rouillard, Barreau 1928	Pluriglandular insufficiency (syphilis ?)	Buccal mucosa gingiva, cheeks tongue, lower lip	Large brown spots or disseminated circles	Not given.
Kleitsch, Korn, Saichek 1955	Intestinal polyposis	Lips, buccal mucosa	Zones of hyperpigmentation	Not given.
Nanta 1926	Leukoplakia on a melanotic lentiginous spot	Mucous membrane of lower lip	Black with irregular white spots sharply circumscribed subjacent melanosis	Intense melanosis constituted by a particularly dense infiltration in the dermis, but also in the basal layer and in the entire height of the epithelium.
Ramel 1925	Lichen planus (3 cases)	Oral mucosa		DOPA positive.
Becker 1927	Lichen planus	Buccal mucosa		Many DOPA-positive dendritic cells in epithelium; dopa-negative chromatophores in connective tissue.
Dummett 1959	Vitiligo Albinism	Oral mucosa Oral mucosa	Depigmented area Depigmented area	Not given. Not given.
Zegarreli et al. 1959	Familial white folded dysplasia 16 patients age 4–42 M & F	Oral mucous membrane	Deeply folded or corrugated oral mucosa with a dense grayish or white keratotic surface	Thickening of all layers of epithelium. Hyperkeratosis. Marked acanthosis with many mitosis. Frequent parakeratosis. Marked swelling and vacuolation in the granular and prickle cell layers.

Fig. 7. Photomicrograph from section of oral mucosa clinically pigmented. Brown-yellow granules are seen in the basal layer and in a few cells of the connective tissue papilla. Thionin. Magnification X190.

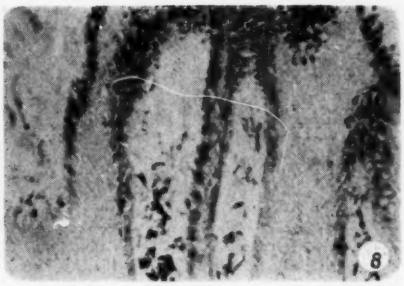

Fig. 8. Higher magnification of an area shown in Fig. 7. Thionin. Magnification X750.

stains mast cells to the exclusion of other cells.[24]

Distinct structural and staining differences in granules of mast cells and granules of melanoblasts from other sites have been shown in other species.[25] No cell was found in mouse skin in which both metachromatic granules (mast cells) and orthochromatic granules (melanin) were contained.[26]

Correlative Findings. Clinical and Histologic. The evidence for the presence of melanin pigmentation in oral tissues is sparse and has been documented from a few discrete observations, and from only a few of the many regions of the oral mucosa (Tables 3, 4, 5, 6, 7).

Grossly unpigmented buccal mucosa from 53 unselected white autopsies were shown by Becker[12] to have slight (epidermal), or marked (connective tissue) pigmentation microscopically. These observations supported the findings of earlier investigators, especially Adachi ('03),[27] who showed dendritic cells in the buccal mucosa of white females.

Ramel ('25)[1] tested the DOPA reaction on sections of twelve buccal mucosae. Six of seven clinically pigmented mucosae were DOPA positive. Five normal non-pigmented mucosae yielded only two that were DOPA negative (Table 3).

Orban and Wentz[28] demonstrated clear cells, melanoblasts, dendritic cells and melanophores in sections of oral mucosa.

Laidlaw and Cahn ('32)[29] demonstrated melanin or melanoblasts or both in sections of clinically non-pigmented gums from Negroes and Caucasians. They reported a high percentage of Negroes showed no oral pigmented areas, and that an equally high percentage of whites did show oral pigmentation. These experiences were recently duplicated, in part, by Breijer and Lignac[30] on a Dutch population (Table 3).

ORAL HYPERPIGMENTATION AND HYPOPIGMENTATION

Increased pigmentation of the oral mucosa is a rather constant clinical finding in a number of systemic disorders. Principal among these are Addison's disease,[28] intestinal polyposis, hemochromatosis, syphilis and oral melanomata. In all the increase is thought to be due to melanin (Table 4).

Addison's Disease. Sections through hyperpigmented spots on both the skin and oral mucosa of white patients showed increase in melanin pigment. Principal sites were in non-dendritic cells and in the basal layer of the epithelium.[31]

Intestinal Polyposis. Intestinal polyposis is associated with cutaneous and oral melanosis.[32] The mechanism of this relationship is not quite clear, but it is thought to represent a congenital defect.[33] Characteristic small round, oval or irregular patches of brown or occasionally black spots extend

TABLE 5.
Histology of Oral Pigmentation—Oral Melanomata

Author Date	*Condition Source Age, Sex*	*Area of Oral Cavity Involved*	*Clinical Status*	*Histology*
Roy 1907	Primary melanosis White 43, M	Palate	Hard brown to black granulations on the whole hard palate with fistula into the nasal cavity	Cells of deeper layers appear to merge into the submuccous tissue. Submucous space is filled with cells of irregular shape, various size and appearance. Spread with no order whatsoever. Melanotic pigment extra and intracellularly.
New, Hansel 1921	Primary melano-epithelioma 62, M	Palate	Tumor one month with rapid growth, pedunculated	Biopsy: (1) melanosarcoma (2) melano-epithelioma.
Orban, Wentz 1955	Pigmented nevi	Gingiva, palate, lining mucosa, tongue	Irregular patches of deep brown, to black discoloration or deeply colored elevated lesions	Proliferation of clear cells with migration into the connective tissue. Nevus cells accumulate as nests and cords surrounded by a connective tissue stroma.
	Lentigo	Gingiva		Enormous increase of melanin pigment in the epithelium and connective tissue with hyperplasia of melanoblasts.
Colby, 1956	Intradermal nevus	Mucous membrane	Enlarging bluish-black or slate-gray raised lesion	Solid sheet of nevus cells in the corium separated from the covering epithelium by a small band of connective tissue.
	Melanoma			Junctional changes in the epithelium. Large atypical malignant melanoblasts in the corium. These may be with or without pigment.
Scoffield 1959	Blue nevus 24, M 41, F White	Hard palate	Bluish pigmented area sharply circumscribed, slightly elevated	Mass of loosely arranged spindle cells in lamina propria. many filled with finely granular dark brown pigment. Also large cells with coarse, densely packed pigment granules.

from the lips onto the buccal mucosa.[34] Biopsy of the oral mucosa from patients who present this syndrome should be subjected to histologic and histochemical analysis in order to determine the true nature of the substances responsible for the melanotic zones observed clinically. From other evidence, it is quite predictable that such study would show increased melanoblasts or increased melanin or both.

Hemochromatosis. In hemochromatosis, a disease resulting from faulty metabolism of iron, there is a distinct bronze discoloration of the oral mucosa,[31] as well as of the skin.[35] In the skin this discoloration is due in part to iron containing pigment deposition in the chromatophores, but primarily to an increase in melanin accumulation or production in the melanoblasts of the epithelium. Sections of oral tissues from patients suffering from hemochromatosis should be subjected to histologic analysis to determine if the same mechanism of discoloration exists.

TABLE 6.

Histology of Oral Pigmentation—Metallic Oral Pigmentation—Embedding of Foreign Objects

Author Date	*Condition Source Age, Sex*	*Area of Oral Cavity Involved*	*Clinical Status*	*Histology*
Goadby 1931	Bismuth pigmentation	Gingiva	Bluish-black throughout	Irregular scattering of bismuth sulphide in subepithelial layer.
Wachstein Zak 1946	Bismuth pigmentation White 55, M	Upper gingival margin	Pigment line	Pigment deposited in superficial capillaries and histiocytes.
	White 59, M	Lower gingiva	Small growth	Same.
Orban, Wentz 1955	Bismuth pigmentation	Gingiva, cheek mucosa, side of tongue	Bluish-black deposits. Small dots or heavy dark lines encircling the teeth.	Fine droplets of black material in the papillary layer of the lamina propria of the free gingiva on the pocket side. Also in the connective tissue or within macrophages.
New, Hansel 1921	Lead pigmentation 40, M	Palate	Small, slightly elevated, blackish hard nodule	Encysted was a small piece of lead 4 mm. long from a lead pencil.
Orban 1949	Lead pigmentation	Gingiva	Bluish-black line around gingival margin	Scattered particles of lead sulphide in the subepithelial layers.
	Graphite 35, F	Gingiva	Bluish discoloration	None.
Irgang, 1936	Gold pigmentation Negro 39, F	Lips, cheeks, soft palate	Discrete black pigmented areas	Not given.
Goadby 1931	Copper absorption	Gingiva	Greenish pigmentation	Not given.
	Carbon pigmentation	Oral mucosa	Bluish mark	Embedded particles of carbon beneath surface of mucous membrane.
Moral, Freiboes 1924	Charcoal impregnation 66, M	Gingiva	Thin lines which run close to and parallel to the gingival margin	Not given. Causes no gingivitis.
Schwartz 1949	Charcoal	Gingiva	Horizontal lines of black pigment	Not given.
Bolden 1959	Carborundum embedding	Lips, mucosa	Bluish-black discoloration	Deposits of discrete black irregular particles in the lamina propria. Typical foreign body reaction.
Cahn, 1932	Silver pigmentation 42, F	Buccal gum	Bluish-black spot 1/8 inch in diameter adjacent to pulpless tooth	Granules of silver in the stroma surrounded by pale staining histiocytes.
Orban 1949	Silver pigmentation (root canal filling) 10, F	Border gingiva and alveolar mucosa	Bluish discoloration	No section.
	Amalgam embedding	Oral mucous membrane, interdental papilla	Bluish-black	Black metallic material embedded deep in the connective tissue within macrophages, around blood vessels, along course of collagenous fiber bundles, giant cells.

Fig. 9. Higher magnification showing a dendritic cell and discrete granules throughout one dendritic process. Thionin. Magnification X1500.

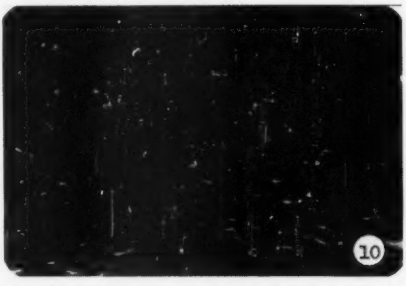

Fig. 10. Photomicrograph showing localization of melanin in basal layer of epithelium of oral mucosa. Masson-Fontana. Mag. X850.

Syphilis. Focal or disseminated round, well limited, black or large brown spots have been reported on the buccal mucosa or gingiva as well as on the skin in local and generalized syphilitic infection.[36-39]

Sections of buccal mucosa and skin taken from a 24 year old syphilitic male showed infiltration by cells with pigment histologically identifiable as melanin. These melanotic changes, especially when accompanying hereditary syphilis have been thought to be due to or to represent pluriglandular insufficiency[39] (Table 4).

Oral Melanomata. Of more serious consequence perhaps are the changes incident to the primary benign and malignant melanomata, lesions which have origin from melanocytes which remain in the deeper connective tissue[28,40-44] (Table 5).

The blue nevus frequently appears on the skin as a sharply circumscribed, smooth, firm, round or slightly elevated papule. Recently two such lesions have been described in the oral cavity.[40] Both arose on the hard palate and were seen in white patients.

Histologically the lesions were composed of spindle shaped melanoblasts arranged in small, irregular bundles in the middle and lower third of the corium and separated from the epithelium by a well defined zone of normal connective tissue. Most of the melanoblasts contained finely granular, dark brown melanin. Varying numbers of chromatophores, which were larger than the melanoblasts, lacked the fine wavy, bipolar processes and contained coarser granules of pigment, were seen in and beside the cell bundles.

Primary melanosis of the palate apparently arising at a site of trauma has been reported.[41] First observed as a small round spot on the median raphe of the palatal vault, the lesion advanced slowly so as to include the entire hard palate with hard, brown and black granulations. Erosion into the nasal cavity was a late feature.

Microscopic examination, 20 years after onset of the original lesion, revealed the submucosa to be filled with cells of irregular shape, various size and appearance, in some places very close to each other; in others they were well separated. Melanotic pigment was seen both intracellularly and extracellularly. These features resembled those of melanotic sarcoma.

Malignant melanoma has been reported in the oral cavity under a variety of names.[43,44] New and Hansel[44] collected 24 cases and added one new case of primary palatal melano-epithelioma. In 19 of the 25 cases no areas of pigmentation were noticed previous to the appearance of the growth. Eighteen of the 25 cases arose in males while seven appeared in females. The average age of the patient was fifty-four years

TABLE 7.
Histology of Oral Pigmentation—Miscellaneous

Author Date	*Condition Source Age, Sex*	*Area of Oral Cavity Involved*	*Clinical Status*	*Histology*
Oard, Kaletsky, Gettinger, 1939	Chronic pulmonary tuberculosis	Oral mucosa	Brownish areas of pigmentation	Undetermined. Probably melanotic. No histology.
	Hemochromatosis	Lips, gums, border of tongue buccal mucosa	Brownish or grayish flecks or diffuse	Hemoglobinogenous elements plus melanin. No histology.
	Pernicious Anemia	Buccal mucosa lips, borders of tongue	Slate colored patches	Undetermined.
	Xeroderma Pigmentosum	Mucosa, cheek	Pigmentation	Not indicated.
	Diabetic Melanoderma	Mucosa, cheek	Pigmentation	Not indicated.
	Phenolphthalein	Mucosa, cheek	Pigmentation	Not indicated.
	Arsenic	Mucosa, cheek	Pigmentation	Not indicated.
	Vagabond's Disease	Mucosa, cheek	Pigmentation, patches of melanosis	Not indicated.
	Irradiation		No change noted	
	Scleroderma	Mucosa of lips tongue, buccal surfaces	Hard, rigid, waxy hyperpigmentation of melanotic type or achroma	Not indicated.
	Acanthosis Nigrans	Tongue, palate gingiva, inner surfaces of lips	Papillary hypertrophy plus melanotic pigmentation	Not indicated.
Keithly, L. 1957	Xanthomatosis	Gingiva	Yellowish-gray discolorations	Not indicated.
	Herpes Zoster	Gingiva	Herpoid patch surrounded by a pigmented area which may become permanent	Not indicated.

with a range from twenty-four to eighty-four years. Generally the lesions grow rapidly and metastasize early.

The true dendritic nature of pigment cells which may be found in such lesions has recently been demonstrated from explants of a malignant melanoma grown in tissue culture (Fig. 11).[42]

Oral Hypopigmentation. Loss of pigment from the oral tissues is recognized principally by loss of color. This is due chiefly to decreases in the amount of melanin (depigmentation) or to surface changes (white patches).

Vitiligo and albinism are characterized by depigmentation of areas of the skin.

Histologically both show the total absence of melanin pigmentation.[45] Recently total oral depigmentation was described as a part of the generalized picture in vitiligo.[46] Sections through the involved oral tissues will probably show features similar to those observed in the skin. Albinism may also have its counterpart in the oral cavity.

White Patches. White patches are observed on the buccal mucosa, the gingiva, the palate, the floor of the mouth, the lip and on the alveolar mucosa. Clinically these may be designated as lichen planus,[1,58] leukoplakia,[47] white folded dysplasia,[57] leukokeratosis,[58] carcinoma-in-situ, or carcinoma.[58] Histologically, the fundamental change directly related to the aberration in pigmentation appears to be an increase in the thickness of the epithelium, and not to changes in the amount of pigment present. The epithelium may show varying degrees of hyperkeratosis, parakeratosis, marked acanthosis, marked swelling and vacuolation in the granular and prickle cell layers, and the derma may show varying degrees of sclerosis.[58]

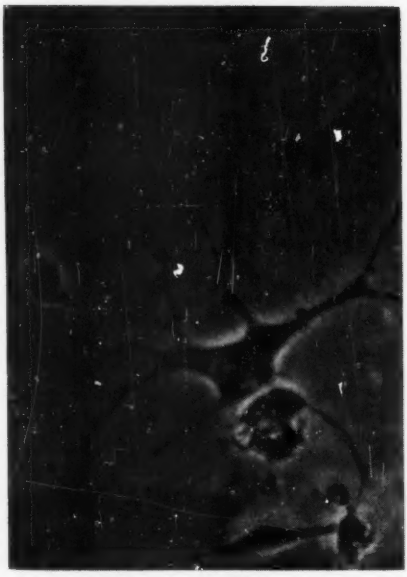

Fig. 11. Photomicrograph of a living dendritic melanocyte of a human melanoma. Unstained. Mag. X845 (Courtesy Dr. Jewel Plummer Cobb).

METALLIC ORAL PIGMENTATION

The metals copper,[48] silver,[49] gold,[51] lead,[44] mercury,[50] and bismuth[48] produce characteristic pigment changes in the oral mucosa due to the accumulation of insoluble precipitates, principally around blood vessels and in macrophages of the subepithelial tissue. Bismuth deposits principally in the pocket side of the free gingiva.[28] Zones of arsenic discoloration and melanotic spots of patients treated with gold sodium thiosulfate showed in addition increased melanin deposition in both basal cells and chromatophores[51] (Table 6).

ACCIDENTAL EMBEDDING OF FOREIGN OBJECTS

Numerous foreign objects may become embedded within the oral tissues and produce localized or discrete zones of pigmentation.[50] Dental amalgam may become dislodged either during placement or tooth extraction, enter the tissues and cause a localized blue-black discoloration.[50] Charcoal used as a dentifrice apparently remained innocuous and produced no tissue reaction other than pigmentation. The patient presented with horizontal lines of black pigmentation, which ran parallel to and close to the gingival margin.[53,54]

Abrasives, such as carborundum, may be introduced into the tissues during operative procedures.[55] Histologic examination through a resected scar from the lip revealed multiple granulomatous nodules within dense collagenous tissue. Pleomorphic masses of black material (carborundum) was found in the center of each nodule and was surrounded by foreign body giant cells, chronic inflammatory cells and variable amounts of dense collagenous tissue. The patient gave a history of having had the lip cut several years previously by a dental stone or disc. Clinically irregular zones of pigmentation had been observed on the mucosa of the lip.

SUMMARY

The histology of oral pigmentation is not unlike that of pigmentation elsewhere in the body, with melanin chiefly responsible. Clinically normal non-pigmented zones have been demonstrated to be rich in pigment cells irrespective of skin color. In addition, certain systemic factors have been shown to influence oral melanin pigmentation.

Several additional pigments and materials have been shown to increase oral pigmentation, though for many, histologic evidence is missing (Tables 3-7).

Important areas in which more data is required include the following:

1. Spectrophotometric analysis of the oral mucous membrane may permit the demonstration of the several pigments and their relative roles in oral pigmentation.

2. Histologic and histochemical analysis of normal oral tissues from various sites would permit mapping of oral pigment quantitatively.

3. Similar analysis of hyper- and hypopigmented zones in albinism and vitiligo should be done as suggested by Dummett.

4. The effect of age on the pattern of oral pigmentation and the relative concentration of pigment producing cells would also prove of inestimable value.

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Diagnosis of Pigmentations of the Oral Tissues*

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A RATIONAL and practical knowledge of the significance of pigmentation of the oral tissues hinges upon: (1) a definition of pigmentation, (2) an appreciation of physiologic pigmentation, and (3) a logical system for considering the various pathologic factors which produce oral pigmentation.

DEFINITION

An examination of almost any medical and lay dictionary[1,2] discloses that the term pigmentation signifies the *deposition of coloring matter*. It follows from this definition that there is a difference between oral *pigmentation* and oral *color*. The color of the teeth and oral mucosa is the hue which results from the deposition of various coloring matters *plus* the color contribution of the subjacent circulating blood in the vascular system. The yardstick to be used in this report will be based on the more restricted definition. In other words, only those conditions will be discussed which result from the *deposition* of coloring matter in the hard or soft tissues of the oral cavity.

Under so-called normal conditions, pigments are present in the oral tissues which contribute to physiologic pigmentation and to the different hues encountered in the mouth. The problem of physiologic pigmentation may be divided conveniently into two categories: (1) the *dental tissues*, and (2) the *soft tissues*.

DENTAL TISSUES

Different teeth and different portions of the same tooth vary in hue because of the relative presence and absence of pigments.[3] For example, the maxillary central incisor is the lightest tooth in the mouth. In other words, this upper anterior tooth approaches white more closely than any of the other teeth in the arch. In addition, the incisal third of the maxillary central incisor is the lightest portion of the crown of the tooth. On the other hand, the cervical third of the tooth is darkest. In general, the maxillary lateral incisor is slightly darker than the central incisor. Also, as in the case of the central incisor, the incisal third is lightest and the cervical third darkest. As one proceeds posteriorly, the teeth become darker and the incisal or morsal third is generally the lightest portion of the crown. The same pattern exists in the lower jaw with the exception that the mandibular lateral incisor is usually slightly lighter than the central incisor. Still, the posterior progression to darkness and the darkening cervicalward on each tooth prevail.

The consensus is that the color of the natural teeth is some shade of yellow due principally to the translucency of the enamel and the pigment content of the dentin. Thus, the incisal third of the maxillary central incisor looks relatively white because there is very little subjacent dentin to contribute the yellow cast and the bulk of enamel incisally is greatest. On the other hand, the thickness of the enamel at or near the neck of the tooth is relatively narrow and there is considerable dentin beneath the thin enamel layer.

SOFT TISSUES

The color of the soft tissues of the mouth is the result of the product of a different set of circumstances. In the first place, the cytoarchitecture of different mucosal areas is quite distinct. In general, three different types of mucosa are present.[3]

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**Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama, on 20 November, 1959.*

Lining Mucosa: The first is strictly a *lining* mucosa. It is very thin, minimally hornified, and very delicate. This is the mucosa which can be observed in areas of the oral cavity which are normally under relatively little mechanical trauma. Thus, one can observe this type of lining mucosa in the buccal area, sublingual space, and the soft palate. In general, lining mucosa is more pink or red than other mucosal zones because of the thinness of the lining wall and the relative nearness of the subjacent vessels to the surface. Color, then, is a function to a large degree of the amount of reduced or oxygenated hemoglobin circulating in the subepithelial blood vessels. Since such color change is not due to the deposition of coloring matter, it will not receive attention in this report. Pigments can and do normally occur in the tissues of the lining mucosa. The most common such pigment is melanin though carotene, caretenoids, and possibly melanoids may also be present.

Masticatory Mucosa: A second type of mucosa is admirably suited to withstand considerable mechanical and chemical trauma.[4] This type is observed in the gingiva and the hard palate. The tissue is characterized principally by a relatively high degree of keratinization. Thus, two points become important. First, the underlying blood vessels are not as close to the surface, hence do not contribute to redness or pinkness as much as in the lining mucosa. Secondly, the keratinization is due to production of eleidin which, in itself, yields a yellow cast. However, pigments can be demonstrated in these tissues. The most common pigment is melanin though carotene, caretenoids, and possibly melanoids may also be present to contribute to physiologic pigmentation.

Specialized Mucosa: The tongue structure is distinctive and quite different from the lining and masticatory mucosae described. Here one encounters on the dorsum of the tongue an area peppered with different types of papillae. The filiform papillae are long and narrow. A large portion of a filiform papilla is taken up by epithelium. Thus, the filiform papillae yield a yellow cast because of the presence of eleidin. In contrast, the second most common projection from the surface of the tongue is the fungiform papilla. This organ is not as heavily keratinized. Therefore, the yellow cast is not as obvious. In addition, because of the relative lack of keratinization, the subjacent blood vessels are apparent. The end result is a projection which appears pink to red in part because of the absence of pigment (eleidin) and partly because of the relatively greater vascular supply. Because of the fewness and posterior position of the circumvallate papilla, they are not significant in any consideration of lingual pigmentation.

Fig. 1. Green stain due to microbial growth.

One other point must be mentioned with regard to pigmentation and specialized mucosal tissue. The distribution of papillae on the dorsum of the tongue allows the entrapment of foreign bodies in the interpapillary spaces. This becomes important because of the greater possibility

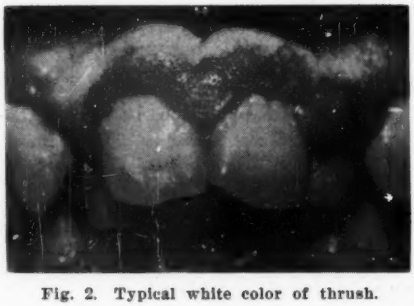

Fig. 2. Typical white color of thrush.

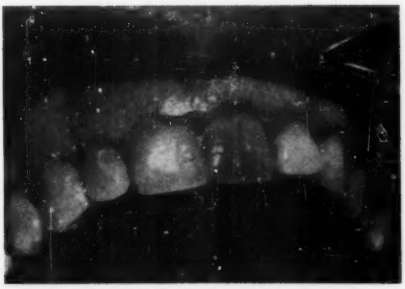

Fig. 3. The darkened left central incisor is due to pulpitis secondary to microbial invasion.

of pigmentary deposition on the dorsum of the tongue as against similar pigmentary incarceration on the lining and masticatory mucosae.

PATHOLOGIC PIGMENTATION

The diagnosis of pathologic pigmentation hinges upon an understanding of: (1) the etiologic factor or factors, and (2) the method by which the pigment reaches the oral tissues.

BIOLOGIC FACTORS

A number of microbial agents can exert an effect upon the dental or soft tissues and produce pigmentation. In addition, these microbes can attack the tissues from the outside (exogenously) or by way of the blood stream (endogenously).

Common examples of pigmentation the result of microbes operating exogenously are the very common *green stain*[5] (Fig. 1) and *orange stain*[6] noted in children. A relatively classic example of pigmentation the result of exogenous microbial action upon soft tissues is observed with *thrush*[7] (Fig. 2).

On the other hand, bacteria, viruses, fungi, and parasites can produce pigmentation of the dental and soft tissues by reaching these structures via the circulating blood stream. For example, the tooth may become discolored following bacterial invasion of the pulp. Thus, we have here an illustration (bacterial pulpitis[8]) the result of endogenous action of biologic factors upon the dental tissues (Fig. 3). In contrast, microbial factors may operate by destroying the vessel wall so that blood is permitted to lodge extravascularly. The end result of extravascular blood deposition leads to decomposition of hemoglobin and the appearance of pigment the result of iron in the tissues. This is the explanation for petechiae in *infectious mononucleosis*[9] (Fig. 4).

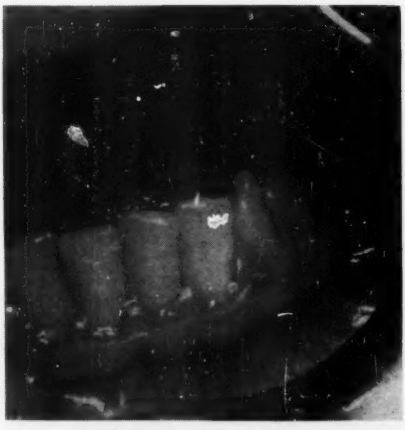

Fig. 4. Typical pigmentary change (petechia) in infectious mononucleosis due to the perivascular deposit of blood.

PHYSICAL FACTORS

Physical factors may take one of several forms including mechanical, electrical, thermal, and radiation trauma. By far, mechanical trauma is most common. Physical trauma may act exogenously or endogenously and the effects may be directed at the dental and/or soft tissues. For example, food particles may lodge on the tooth surface or on the soft tissues and may remain for a variable period of time depending on the speed with which cleansing occurs. This type of pigmentation does not produce a diagnostic problem since the etiology is relatively clearcut and the therapy is simple and often unnecessary. On the other hand, endogenous influences may be operative so that the end result is pigmentation due to some type of physical trauma. This is well illustrated in *mechanical perice-*

Fig. 5. Contusion to the lip causing the extravasation of blood into regional tissues and eventual decomposition of blood products.

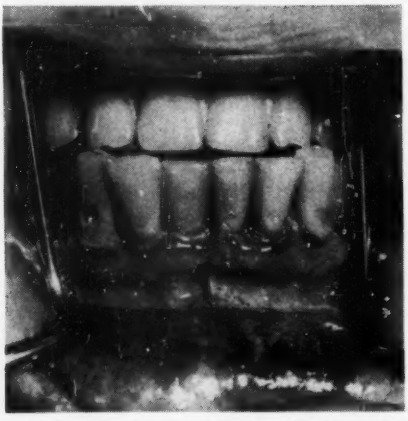

Fig. 7. Mercurialism with metallic gingival pigmentation

mentitis.[10] This is a situation where a mechanical insult is brought to bear upon a tooth surface so that there is eventual pulpal death and alteration in the pulp and dentine because of the absence of nourishment to the tooth. The very simple and obvious example of the results of physical trauma upon the soft tissues is shown in the contusion (Fig. 5). Here the situation is one in which physical trauma is brought to bear upon the soft tissues so that small blood vessels are caused to rupture. The blood which lodges extravascularly then undergoes decomposition. First the area appears red, then purple, then black and blue, and finally the area clears up through a progression of color changes leading through brown and green.

CHEMICAL FACTORS

A host of chemical substances can exert an effect upon the dental or soft tissues exogenously or endogenously and in this manner lead to pigmentation. Certainly, *tobacco stain* is a common illustration to show the deposition of various fractions from tobacco upon the tooth surface and the neighboring soft tissues. However, as has been previously mentioned, it is difficult to delimit exogenous or endogenous factors for, in many cases, both are operative. For example, the systemic intake of fluorides in the drinking water may pro-

Fig. 6. The exogenous pigmentation associated with classical fluorosis made possible by the changes in the enamel surface.

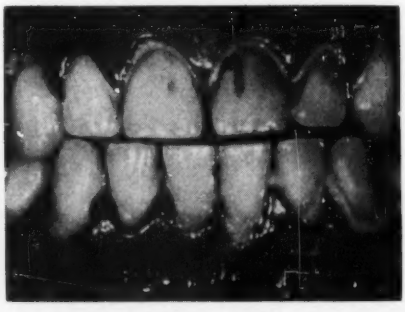

Fig. 8. Brown pigmentation of the oral mucosa (melanosis) due to hypoadrenalism.

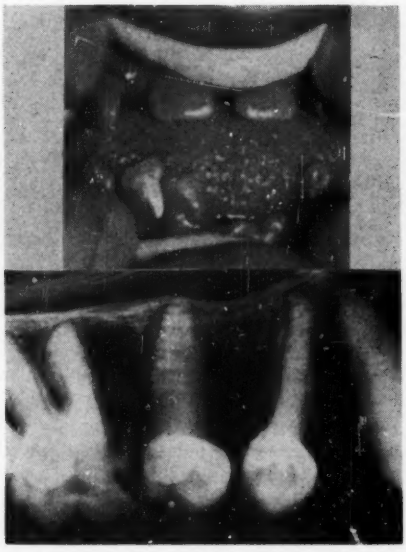

Fig. 9. Hereditary opalescent dentin in a 10 year old boy with osteogenesis imperfecta. The crowns are disproportionately large for the roots and the pulp chambers and canals are obliterated.

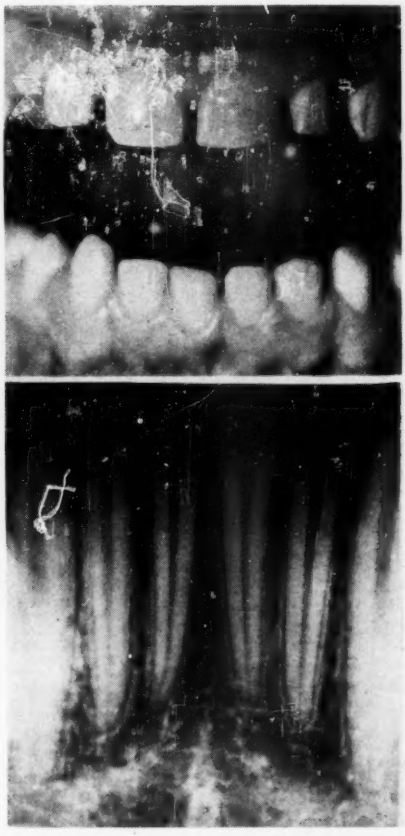

Fig. 10. The teeth in amelogenesis imperfecta (enamel hypoplasia type) are yellow because of the relative absence of enamel. Shown in the roentgenograms is the absence of enamel roentgenopacity.

duce changes in the tooth surface characterized by small chalky areas.[11] However, because of the alteration in the tooth surface, exogenous materials find it more easy to lodge upon the tooth. Thus, the pigmentary changes described with fluorosis actually are the result of exogenous deposits upon a tooth surface which allows such deposits to lodge easily (Fig. 6). Occasionally foreign bodies are present in the soft tissues and, in their disintegration, color the neighboring structures. This is commonly encountered in the situation whereby a small fragment of amalgam is trapped in the oral soft tissues.

On the other hand, endogenous factors may play a more dominant role than has been shown, for example, in the case of fluorosis. For instance, chemical pulpitis[12] may follow pulpal death due to a silicate restoration which has been placed in a tooth without appropriate protection of the subjacent tissues. Similarly, endogenous factors may play a role in the development of pigmentation of the soft tissues. For instance, many cardiac patients are required to take mercurial diuretics. This, of course, means that mercury is circulating in the blood stream. In areas of inflammation hydrogen sulfide is formed. The end result is that the mercury unites with hydrogen sulfide to produce mercuric sulfide. This condition, *mercurialism*,[13] is commonly encountered in the free marginal gingiva (Fig. 7). This is also believed to be the explanation for the pigmentary changes in the gingiva in patients with plumbism.[14]

Fig. 11. Melanin spots characteristic of intestinal polyposis.

Fig. 12. Amelogenesis imperfecta (enamel hypocalcification type). The pigment is exogenous.

HORMONAL FACTORS

Mention has already been made that each group of factors may operate exogenously and endogenously. It would, therefore, seem that these two routes must be operative in the case of hormonal imbalance. However, little study has been directed to the effect of exogenous hormonal factors upon dental and soft tissues. Certainly, the effect is clearcut that there is deepening of color of the skin and mucosae in patients with hyperpituitarism[15] and hypoadrenalism.[15] The classical descriptions of so-called bronzing of the skin are described with *Addison's disease* (Fig. 8).

DEVELOPMENTAL FACTORS

Developmental factors are conditions in which alterations occur in the oral tissues either because of an inheritable factor or because of changes in the developing fetus during intrauterine maturation. Developmental disorders may be accompanied by oral pigmentation for two reasons. In the first place, the oral tissues may be malformed in such a way as to produce abnormal pigmentary patterns. This is well illustrated by the *hereditary opalescent dentin*[16] with or without osteogenesis imperfecta (Fig. 9). In this instance, the chemistry of the dentin is so altered that, in combination with the usual translucency of enamel, the dental tissues appear opalescent. Amelogenesis imperfecta[17] of the *enamel hypoplasia type* also produces an unusual pigmentation of the teeth. In this instance, it is due to the inadequate production of enamel so that the underlying dentinal color is more apparent. Consequently, persons with this problem show unusually yellow teeth (Fig. 10). An example of soft tissue pigmentation of developmental origin is well illustrated in intestinal polyposis[18] (Fig. 11). Here one encounters macular lesions in the oral cavity which actually represent discrete depositions of melanin.

On the other hand, the developmental factor may so alter the hard or soft tissues of the oral cavity so as to invite exogenous stains to deposit more easily. This is illustrated in *amelogenesis imperfecta*[17] (Fig. 12) of the enamel hypocalcification type. Here the problem is not one of adequate enamel matrix formation but one of imperfect calcification. Consequently, the enamel is soft and is lost from the tooth very easily. The end result is a roughened surface which invites the deposition of outside stains.

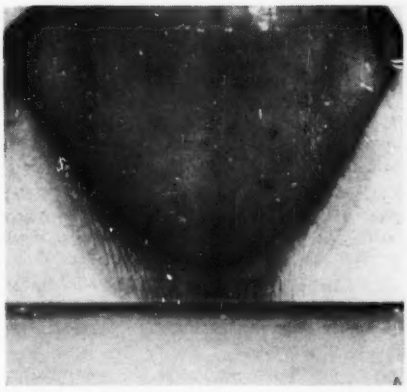

Fig. 13. Petechiae in the palate due to increased capillary fragility and permeability secondary to Vitamin C deficiency.

Fig. 14. Rachetic teeth. The exogenous pigmentation is possible because of the roughened tooth surface.

NUTRITIONAL FACTORS

Nutritional imbalance is apt to be associated with pigmentary alterations of the soft tissues for two reasons. In the first place, the absence of certain nutrients from the dental tissues may so alter the chemistry of the enamel and dentine as to yield a tooth of unusual color. Also, vascular fragility and permeability are very much dependent upon nutritional factors such as ascorbic acid[19] (Fig. 13). Consequently, with increased vascular fragility and permeability the extravascular appearance of blood is very possible. The end results are petechiae and/or ecchymoses.

Nutritional factors play an important role in normal maturation of oral tissues. Consequently, with nutritional imbalance during the formative years of dental development the end result may lead to hypoplasias. These roughened surfaces permit the more ready deposition of exogenous pigment. This is demonstrated in rickets[20] (Fig. 14).

STRESS DISORDERS

Every local stimulus is accompanied by a local response. However, every local excitory force is also associated with a generalized reaction.[21] This systemic response is important to the organism encountering the local stressor. Should the generalized reaction not occur, then serious complications may ensue. Thus, more correctly, stress disorders are really disorders due to lack of adaptation. Any serious stressor during dental development may leave its mark upon the forming dental tissues. For example, high fever in a child may be recorded permanently in the dental apparatus by way of pits, fissures, or other evidences of hypoplasia. These roughened areas make it more possible for exogenous stains to attach themselves to the tooth structures. One of the most important factors in the maintenance of vascular fragility and permeability is adrenocortical balance. In disorders due to lack of adaptation to stress, hypoadrenalism is present. Thus, with reduced adrenal activity there is the possibility of increased vascular fragility and permeability. Consequently, petechiae and ecchymoses may be present.

SUMMARY

1. Strictly speaking, *pigmentation* signifies the *deposition* of coloring matter in a tissue. In contrast, *hue* represents the color of a tissue which is due to pigmentation and the status of the subjacent vascular supply.

2. Under normal conditions, different teeth and different portions of the same tooth differ in hue because of the thick-

ness of the enamel and the character of the subjacent dentin.

3. Under physiologic conditions, different portions of the oral mucosa differ in color in part because of various pigments present in the tissues and also because of the blood supply.

4. In some instances, *biologic, physical, chemical, hormonal, developmental, nutritional,* and *stress* factors may be singly responsible for the presence of pathologic pigmentation. These seven factors may operate by *exogenous* or *endogenous* mechanisms.

6. In most cases, pathologic pigmentation is the result of a combination of circumstances operating both exogenously and endogenously.

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The Melanomata*

by J. C. ASHHURST, M.D.,** *Tuskegee, Alabama*

Definition — Melanomata are tumors composed of cells having the potentiality of forming melanin.

Classification

A. Benign melanoma—pigmented nevi of skin. Intradermal (common moles) and junctional nevi (potentially malignant) are distinguished.

B. Pigmented nevus (birthmark or mole):
   1. Congenital in origin.
   2. Nevus is of neural origin (Masson).
   3. Found on any part of the body.
   4. Varies in color from gray to black.
   5. Grows slowly.
   6. Irritation more frequent when lesion is raised above surface.
   7. Variable amount of pigment not related to malignancy.

C. Malignant nevus—early metastasis:
   1. Regional lymphadenopathy.
   2. Metastasis also via blood stream to involve large organs and bones.

Melanoma

A. Tumors composed of potential melanin-forming cells.
   1. Cells are dopa positive.

B. Malignant melanoma
   1. Superficial type
      a. Reaction confined to junctional portion of epidermis.
      b. No invasion into the dermis.
      c. Prognosis: Chances good for five year survival.
   2. Malignant type
      a. Invasion of the dermis.
      b. Prognosis: Hopeless.

C. Oral melanoma
   1. Melanomas also occur in the oral cavity forming from the epithelium.
   2. Intraoral melanoma derived from melanoblasts found in pigmented areas as well as in adjoining uncolored parts of mucosa of human gingiva, cheek, palate, pharynx and floor of mouth.
   3. Oral melanoma is extremely rare.
   4. Histopathology.
      a. Intraoral melanoma is similar histologically to malignant mole.
      b. Melanoblasts vary in shape and arrangement, occurring as fusiform or round cells.
      c. Stroma delicate and scarce.

D. Etiology of malignant melanoma
   1. Reasons are not specifically known, but there are many hypotheses.
   2. Trauma—tendency to discount role of trauma in pathogenesis (Allen).
   3. Irradiation and sunburn—malignant melanomata have arisen in scars and burns. The possibility of ultraviolet radiation has been considered.
   4. Humoral factor.
   5. Neoplasms behave poorly in pregnant women, are relatively benign in children and older age groups.
   6. Administration of ACTH—activation of junctional nevi (Allen).

E. Incidence of melanoma
   1. Malignant melanoma ranks third in its propensity for intracranial metastasis.
   2. Allen reported a ratio of whites to Negroes at 4 to 1 (900 cases).
   3. In 400 cases reported from South Africa, incidence of malignant melanoma was 2.2 per cent (whites), 3.38 per cent (Negroes).
   4. Though rare, malignant melanoma does occur in Negroes. These tumors occur with the same frequency as brain tumors.

**Acting Chief, Laboratory Services, VA Hospital, Tuskegee, Ala.

**Outline of presentation given at the First Symposium on Oral Pigmentation, November 20, 1959.*

Areas of Research in Oral Pigmentation*

by C. O. DUMMETT,[1] D.D.S. AND C. T. MASON,[2] PH.D., *Tuskegee, Alabama*

HAVING explored much of the available information on the subject of pigmentation of the oral tissues in such detail, it is now possible to see that comparatively little is known, and much needs to be investigated on this interesting subject. Such information can be obtained only by extensive research activities in these areas. With the great emphasis now being placed upon the pursuits of knowledge in so many aspects of human endeavor, it should not appear unusual to expect that similar emphasis needs to be placed on this aspect of human biology.

The sporadic and isolated instances of scientific thought and exploration on this problem, have pointed out the fact that there is an urgent and definite need for unified action by scientists—the dentist, physician, biologist, chemist, veterinarian, histologist, pathologist, and others—if the problems of pigmentation are to be solved. Acknowledging the debatability as to whether or not there is the need to bring the situation to public attention, one cannot question the observation that a subject which has any relationship to human physiology and pathology, and to human psychology and sociology, should be fully explored. Accurate, truthful knowledge should then be made available to the public.

This exploration, this pursuit of knowledge requires financial assistance, so that it is not too unrealistic to suggest the inauguration of some financial support for research in oral pigmentation. A fund created for such a purpose would join the many other vital funds and foundations which, through the years, have been proposed and have served so well to regulate and to structure the financial subsidizing of new knowledge.

The listing of specific research projects is not the aim of this presentation. What will be attempted is an identification of general areas which appear to merit further consideration and study. These might be grouped into 3 convenient categories—

1. basic research in oral pigmentation
2. clinical research in oral pigmentation
3. research in the psycho-social aspects of general pigmentation with special considerations of oral pigmentation.

BASIC RESEARCH

Melanin is believed to be the substance imparting pigmentation in plant, animal, and insect life. A fundamental study of this substance, leading to a thorough and complete knowledge of its preparation and properties, is essential to basic research on pigmentation. A starting point in this research might well be the making and publishing of a review on melanin by a well-qualified scientist. While there are reviews now in the literature they seem to be in obscure and not easily accessible journals. Such a review as is proposed, could be important in developing interest in melanin research by allowing the research worker to view the extent of present knowledge and, more importantly, to chart future work having the best possibility of leading to fruitful results. Further, such a review would focus attention on the subject and serve to spark and encourage new workers in the field.

From the literature consulted, it seems that there is doubt concerning the structure of melanin. The melanins isolated from various natural sources could be different not only from each other, but also from that prepared in the laboratory. The isolation, preparation, and characterization of the melanins from these various sources

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*Presented at the First Symposium on Oral Pigmentation, Veterans Administration Hospital, Tuskegee, Alabama, on 20 November, 1959.

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could be an important field for biochemical research. The methods of physical and organic chemistry might well be applied in establishing properties of melanin, and of compounds similar in structure to its precursors. This could lead to information helpful to further research.

Significant research can emanate from biochemical studies on the relationship of light, sulfhydryl groups, hydrogen ion concentration, copper and other heavy metal salts, oxygen, carcinogenic agents, the various amino acids, and other chemicals which are known to affect melanin formation. Furthermore, the effects of nutrition on melanogenesis, of protein on protein-free melanin, of the pituitary hormones, and of the various fungi causing skin discolorations—all can lead to an understanding of skin pigmentation adaptable to oral pigmentation. Truly, melanogenesis is a fertile field for basic research.

CLINICAL RESEARCH

Insofar as clinical research in oral pigmentation is concerned, it would appear that there is a need for information about the clinical findings and their causation in disturbances of pigment formation. These disturbances include:

(a) *Hyperpigmentation* — The reasons behind excessive pigmentation under normal circumstances need to be investigated. This problem is of course closely related to the mechanism of pigmentation per se. When pathological conditions enter the picture, these pigment disturbances take on added significance.

(b) *Hypopigmentation* — The lessened clinical presence of pigment in oral areas is another question which should be explored.

(c) *Apigmentation*—There is a need to know much more about the hereditary absence of melanin pigment, usually referred to as albinism. The epidemiology of albinism as well as the incidence of certain collagen diseases in albinos are other important areas which should be investigated.

(d) *Depigmentation*—Vitiligo is probably the most familiar of the diseases which produce a loss of pigment in areas previously pigmented. Details of the immediate and long range effects on the oral tissues need to be worked out. The neural influence in melanin pigmentation is said to be closely related to depigmentation, and should be investigated.

(e) *Repigmentation* — There are in progress some studies by the senior author to determine the time necessary for repigmentation to occur in gingivectomized tissues.

A few other areas in need of investigation include:

(1) epidemiological studies on the racial differences in the concentration and activity of oral melanocytes, (2) the hormonal and neurogenic control of melanin pigmentation, (3) the etiology of oral color changes in pregnancy, cirrhosis, the avitaminoses and ageing, (4) the viruses and oral pigment, (5) pigmentations in oral neoplasms, (6) endocrinology and oral pigmentation.

RESEARCH IN PSYCHO-SOCIAL ASPECTS

It is a generally acknowledged fact that there are psychological and social reactions to the clinical existence of general skin pigmentation. Dental clinicians can attest to the existence of equally insistent psychosocial reactions to the clinical presence of melanin in the oral tissues.

The specificity of these reactions, their significance, their etiology and classification have not been explored.

This is an area wherein conjecture and superstition have been allowed unrestricted influence and have swayed general opinions. These and other irrational reactions would seem to indicate the existence of a virgin field of investigation for some enterprising psychologist, social scientist or psycho-socially-motivated dentist. It is to be hoped that in the near future there would be a representative number of investigations in all of the categories suggested.

Patient Reactions to Brushing Teeth with Water, Dentifrice, or Salt and Soda*

by NANCY J. DUDDING, R.D.H.; LILLIAN O. DAHL, R.D.H., AND JOSEPH C. MUHLER, D.D.S., PH.D., *Bloomington, Indiana*

IT is a generally accepted fact in dentistry that proper and regular toothbrushing is associated with not only less dental caries, but better gingival health. Clinicians are well aware of the fact that these observations are more dramatic in patients who previously practiced poor oral hygiene and then were able to improve their habits and practice good oral hygiene. It is equally well considered to be a proven fact by some dental authorities that the toothbrush alone is the item responsible for producing these biological effects and that the use of a dentifrice serves only to make brushing more pleasant, but is without value in improving oral health. No studies have been reported, to our knowledge, which evaluate these effects when no dentifrice was used, thus making it impossible to separate the effects of the dentifrice and the toothbrush. It was, therefore, considered important to test the anticariogenic effects of good toothbrushing techniques in the absence of a dentifrice. Such a study was planned and designed using techniques previously described by us[1] for similar dental caries tests when evaluating a dentifrice. However, when the subjects were selected and placed in the program, those who were to brush daily with no dentifrice would not remain in the program for over a 6 week period, thus making it impossible to estimate dental caries incidence. The subjects' reactions to this program were so dramatic that it was considered significant to evaluate them and to extend the findings to an evaluation of the subjects' reactions to a dentifrice, and to salt and soda.

EXPERIMENTAL PROCEDURE

A group of college students was asked to participate in a study in order to determine their reactions to brushing their teeth with water alone as against brushing with a paste or a powder dentifrice. Approximately 280 students were asked before 50 were found who were willing to participate in the program. The study consisted of three parts. Part I was a determination of the subjects' reactions when only a toothbrush was used. Part II was a determination of the reaction of the same subjects who then were given a dentifrice composed of salt and soda.

Each student who agreed to participate was given a thorough dental prophylaxis at the initial visit. Following the prophylaxis, the labials of the anterior teeth were photographed using Eastman Kodak color film. A disclosing solution† was then applied and the patient was allowed to rinse lightly with water and the teeth were rephotographed. Additional photographs were made after 2 and 5 weeks in order to have permanent records for classification. In this portion of the study an estimate of pellicle formation was made. Pellicle was classified according to color, severity, position on the teeth, and number of teeth affected by drawing the affected areas on tooth charts with appropriate color crayons. For purposes of recording, an arbitrary classification based upon severity of stain, affecting at least the four maxillary and four mandibular anterior incisors was required before an individual case was reported as having pellicle present. For Part I of this study, each patient was dismissed

Indiana University Dental Study Unit.

*This study was supported in part by a grant from The Procter and Gamble Company, Cincinnati, Ohio.

†Composition of the disclosing solution was as follows: Iodine, 50 parts; Potassium iodide, 15 parts; Zinc iodide, 15 parts; Glycerine, 240 parts; and, distilled water, 240 parts.

TABLE I
Replies (in percentages) to Questions Asked Subjects in the Three Study Groups

Study Group	*Question No.*	*Yes*	*No*	*Indifferent*	*Undecided*	*Withdrew*
I	a	0	44	16	2	38
	b	14	48	—	—	38
	c	38	24	—	—	38
	d	6	42	14	—	38
	e*	—	—	—	—	—
	f	4	40	10	—	46
	g	4	42	0	8	46
II	a	88	0	7	—	5
III	a	8	31	41	—	20

*In answer to this question 8% said they brushed longer; 16% brushed more carefully; 14% brushed more carefully and longer; 8% brushed more thoroughly; 12% brushed same as before; 4% brushed for a shorter time; and, 38% withdrew.

and instructed to brush his teeth twice a day using only water and a toothbrush and to use no dentifrice or mouthwash. Toothbrushes were supplied so that the quality of the brush would be uniform for all participating patients.

After 2 weeks, the participants were recalled, re-photographed, and classified for pellicle formation. The following questions were asked of each participant:

Part I. *Evaluation of Brushing Teeth Without Dentifrice.*

Question a: *Do you like using no dentifrice?*

Question b: *Does your mouth feel clean using no dentifrice?*

Question c: *Do your teeth feel clean using no dentifrice?*

Question d: *Would you consider continuing forever to brush without a dentifrice?*

Question e: *Do you brush your teeth differently when you use no dentifrice?*

At the end of five weeks, the participants were asked the following questions:

Question f: *Do you like using no dentifrice?*

Question g: *Would you consider using only a mouthwash and not brushing your teeth at all?*

Part II of the study consisted of giving the participants another prophylaxis, toothbrushes, and a non-fluoride paste dentifrice. They were instructed to brush their teeth twice a day and use no mouthwashes. After 2 weeks and again after five weeks, the participants were re-photographed for pellicle formation following the same procedure as in Part I. At the completion of the second part of the study, the following information was collected:

Question II a: *Do you like using toothpaste?*

Before the third part of the study the participants were given a prophylaxis and supplied with toothbrushes and a powder dentifrice composed of equal parts of table salt and baking soda. They were instructed at this time to brush their teeth twice a day and use no mouthwashes. After two weeks, the participants were photographed as in Parts I and II. At the conclusion of Part III the participants were asked:

Question III a: *Do you like using tooth powder?*

Question b: *List in order of your preference: no dentifrice, paste dentifrice, and powder dentifrice.*

Question c: *List three things, in order of importance, that motivate you to brush your teeth.*

TABLE II

Group	*Time in Weeks*	*No. of Subjects*	*No. of Subjects with Pellicle Formation*	*Percentage with Pellicle Formation*
No dentifrice	2	31	12	61
	5	27	25	93
Dentifrice	2	47	3	6
	5	47	4	9
Powder	2	40	4	10

DATA AND DISCUSSION

The data in this study are found in Tables I through III. The data in Table I show that by the end of 14 days, 38 per cent of the subjects in Group I had withdrawn from the study since they refused to continue in the program if they had to use no dentifrice (I a). By the end of five weeks, 46 per cent of the subjects had withdrawn (I f). Furthermore, an additional 40 per cent of the subjects expressed a strong dislike for using no dentifrice. Only 4 per cent of the group stated that they were in favor of this procedure. These data similarly confirm much previous information from this clinic indicating that considerable numbers of people will not brush their teeth if they cannot use a dentifrice. Such findings suggest that an acceptable dentifrice must be used before the majority of people will clean their teeth. If less dental caries is associated with the routine use of the toothbrush it seems impossible, as judged from these data, to affect a caries reduction without the use of a dentifrice. The philosophy that "the toothbrush" is the only important factor seems to be unwarranted since without an acceptable dentifrice most patients failed to clean their teeth. This can be demonstrated by an examination of Illustrations 1 and 2. Illustration I a is a photograph of a patient at the initial visit immediately following a thorough prophylaxis while I b is the same subjects following an application of the disclosing solution. Illustration 2 a is the same subject after 2 weeks who brushed his teeth with a toothbrush, but without the use of any dentifrice. The pellicle formation is evident. This material was quite difficult to remove and was tenaciously held to the teeth. Illustration 2 b is the same subject at the end of 2 weeks and to whom the disclosing solution has been applied. One notes that even though the subject used the toothbrush the teeth still contained noticeable amounts of materia alba. This case as well as similar ones in this study show quite dramatically that the use of the toothbrush without a dentifrice will result in many instances, in the subject's failure to clean his teeth properly.

Table II shows the effect of the three oral hygiene procedures on pellicle formation. The groups which used no dentifrice had the highest incidence of pellicle formation. After as short a period as 2 weeks 61 per cent of the subjects had pellicle formation of such a severity as to fulfill the requisite for classification. At the time of the 5 week period, 93 per cent had pellicle formation. In 55 per cent of these cases 74 per cent of the labial or buccal surfaces of all teeth were affected. The group using the dentifrice had measurable pellicle after 2 weeks, but this involved only 6 per cent of the sample. Similar findings were found for the group using the soda-salt dentifrice powder. These data provide additional evidence in support of the wisdom of recommending an acceptable dentifrice in order to encourage patients to brush their teeth. Evidence of pellicle formation is sufficient justification to patients (73 per cent of the subjects using no dentifrice specifically objected to this pigmentation) to keep them from using only a toothbrush to clean their teeth. These data, coupled with the fact that few people brush their teeth with

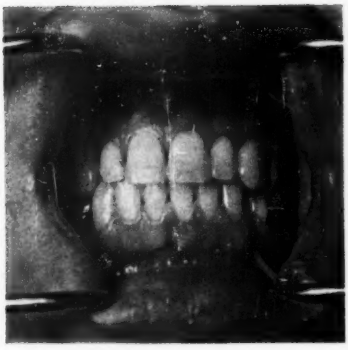

FIGURE 1a. Photograph of a patient at the initial visit immediately following a thorough prophylaxis.

FIGURE 1b. Same subject following an application of a disclosing solution.

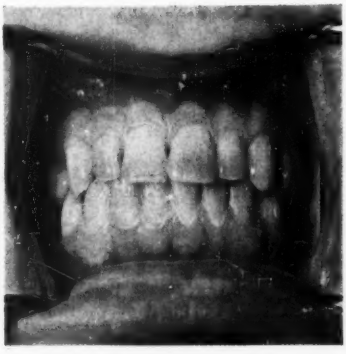

FIGURE 2a. Same subject, who brushed his teeth with a tooth brush without any dentifrice for two weeks. Note the pellicle formation which is difficult to remove.

FIGURE 2b. Same subject, after a disclosing solution has been applied. Note that even though the subject used a tooth brush the teeth still contained noticeable amounts of materia alba.

sufficient frequency[2,3] to reduce caries with a high degree of significance by this method would suggest that one way of providing better oral hygiene would be to encourage the use of an acceptable dentifrice.

While 38 per cent of the subjects using no dentifrice said that their teeth felt clean, 48 per cent reported that they did not. When asked if they would consider continuing to brush without a dentifrice, only six per cent of the subjects reported affirmatively (I d). Only four per cent of the subjects using no dentifrice said they would consider using only a mouthwash and not brushing their teeth at all.

A total of fifty per cent of the Group I participants stated that they brushed their teeth differently when they used no dentifrice. Of these, four per cent said they brushed for a shorter time, forty-six per cent of the subjects said they brushed either longer, more carefully, or more thoroughly in order to compensate for using no dentifrice. However, the data in Table II indicate that they did a poorer job of cleaning their teeth (see Illustration II also). More people complained of their mouth "not feeling clean." They expressed concern about the bad taste in their mouth and were obviously worried about an unpleasant mouth odor, more so, in fact, than about their teeth not being clean.

At the end of five weeks, only five per cent of the subjects in Group II (the group using paste dentifrice) had withdrawn from the study and eighty-eight per cent stated that they liked using toothpaste. This is in contrast to 46 per cent who withdrew at this same period when they used no dentifrice. No one expressed a dislike for this part of the study.

The largest percentage (41 per cent) of the Group III participants (those using "salt and soda" powder), were indifferent when asked if they liked the powder. Twenty per cent of the subjects withdrew from this part of the study and an additional 31 per cent disliked using the powder.

TABLE III

Question III b: *List in order of your preference: no dentifrice, paste dentifrice, and powder dentifrice*

Paste-powder-water	59%
Paste-water-powder	13%
Powder-paste-water	5%
Undecided	3%
Withdrew	20%

Question III c: *List three things, in order of importance, that motivate you to brush your teeth.*

	Total	*I*	*II*	*III*
Cleanliness	18%	25%	23%	6%
Breath	14%	6%	31%	3%
Habit	12%	6%	12%	17%
Appearance	7%	6%	6%	10%
Taste	17%	23%	6%	23%
Health of Teeth	20%	28%	16%	16%
Social Pressure	5%	3%	6%	6%
Obligation to Study	2%	3%	0%	3%
Didn't know	5%	0%	0%	16%

When asked to list their order of preference for the three groups at the conclusion of the study, 59 per cent listed paste first, powder second, and no dentifrice last; 13 per cent listed paste first and powder last. Only five per cent listed powder first and paste second.

Table III shows the replies given when the participants, at the conclusion of Part III, were asked to list their motivations for toothbrushing. It is gratifying to note that these students listed more frequently (20 per cent) the "health of the teeth" as their motivation for brushing their teeth. It is encouraging to find that the efforts of the dental profession are making an impression in educating the public to the need for maintaining good oral hygiene and its relation to mouth health. Children in another study[4] who had "good" oral hygiene said they learned this practice from the family dentist. "Habit" was given by 12 per cent as their reason for brushing, so this would emphasize the importance of teaching regular brushing habits at an early age. It was interesting to note that only seven per cent of the total listed "appearance" as the motivating power for brushing.

SUMMARY

A group of 50 college students participated in a study in order to determine their reactions to brushing their teeth without a dentifrice, with a paste dentifrice, and with a powder dentifrice. Although the total number of participants was small, the findings seemed significant. The general reaction of the participants who used no dentifrice was most unfavorable. This is indicated by the fact that 46 per cent of the subjects withdrew and only 4 per cent stated that they liked to brush without a dentifrice. By contrast, in the group using a paste dentifrice, 88 per cent of the participants stated that they preferred this procedure and only 5 per cent withdrew. When the participants were given a "salt and soda" powder, 20 per cent withdrew, and 8 per cent said that they liked using the powder. Pellicle formation was found in 61 per cent of the subjects using no dentifrice, in contrast to only 6 per cent using a paste dentifrice and 10 per cent using a powder dentifrice after a two weeks evaluation period.

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AMERICAN ACADEMY OF DENTAL MEDICINE

The American Academy of Dental Medicine will conduct its 1961 annual meeting in the form of a cruise convention to Bermuda and Nassau aboard the luxury liner S.S. Ocean Monarch from Friday, May 26 to Saturday, June 3, it has been announced by Dr. Howard L. Ward, chairman, and Dr. Henry Greene, co-chairman.

The scientific discussion will feature the topic "Recent Trends in Dental Medicine," with outstanding clinicians presenting their findings. A joint meeting with the Bermuda Dental Society at that island community will feature the program with Dr. Hermann Becks, Academy president, as a speaker.

Members are urged to make reservations early by sending a deposit of $100 to Travel Guide Agency, attention of Mr. Eugene Fisher, 416 North Charles Street, Baltimore, Md.

Registration fee for the scientific session is $10. Checks should be made payable to Dr. Rudy Palermo, 106 Central Avenue, Brooklyn, N. Y.

New Concepts in Splinting the Mandibular Anterior Teeth

by GLEN H. CURTIS, D.D.S., M.S.,* AND LLOYD BAUM, D.M.D., M.S.,**
Loma Linda, California

THE finding in an Egyptian tomb of two teeth ligated by a gold wire is evidence that the treatment of periodontal disease and an attempt to save loose teeth has occupied the attention of those endeavoring to treat the oral cavity since the dawn of recorded history.[1]

Periodontal splinting may be accomplished in various ways, the use of cast gold restorations providing the most refined and permanent way. With the advent of power-driven, rotary-cutting instruments, the problem of removing tooth substance in preparation was made easier; but in cases where multiple preparations were desired, the problem of making parallel grooves to obtain a line of "draw" for the wax pattern has been a most difficult task. The nature of the oral cavity and the limitation of space in which to work have made it difficult to use mechanical devices for obtaining parallelism in cavity preparation. Nevertheless, an intra-oral paralleling technique, which was developed by Karlstrom[2] of Sweden, has been in use over the past 20 years. Despite the lack of newer impression materials, Dr. Karlstrom's Ponto-Structor Method[2] has produced long-span bridges and other reconstruction work which have been in service for many years.

It is felt by many periodontists that full veneer coverage for the purpose of splinting is a radical procedure. In addition, the procedure is time consuming and costly, thereby limiting its application. The technique to be described here is the result of research at the College of Medical Evangelists, Loma Linda, California. It is a unique and simplified method which enables one to fabricate, in a brief period of time, a cast gold splint for the six mandibular incisor teeth by means of parallel pins—a method which conserves tooth structure and augments esthetics. Advantages of this technique are: (1) Conservation of tooth structure. (2) Positive strong retention. (3) Improved esthetics.

TECHNIQUE

This technique centers around the use of some uniquely different equipment and supplies, particularly with respect to preparing the multiple miniature pinholes.

The drilling of the pinholes is done with a specially prepared jeweler's drill[3] mounted in a back-action handpiece.

It is not intended that the drill fit rigidly within the handpiece; a loose and wobbly fit is necessary. This permits freedom of motion as needed.

The alignment of all the pinholes in a parallel direction is done with a special aligning jig[3] which is mounted to the labial surfaces of the mandibular incisor teeth with impression compound. The drill bushing, which is on the end of the terminal arm of the swivel, will guide the drill in a given direction regardless of its location in extension or rotation. The central axle will permit the bushing to be moved lingually or labially to conform to the curvature of the arch. As shown in the illustrations (Figures 3 and 4), the drill is passed through the drill bushing and the back action of the handpiece permits the drill to penetrate in a labial direction. The bushing provides a firm, rigid path of alignment while the handpiece, acting as a universal joint, provides the power for the drill. This principle and design of the drill adaptor for the handpiece are incorporated in Karlstrom's Ponto-Structor Method.[2]

Not all teeth are suitable for splinting

*Assistant Professor Periodontics, and **Associate Professor Restorative Dentistry, School of Dentistry, College of Medical Evangelists, Loma Linda, Calif.

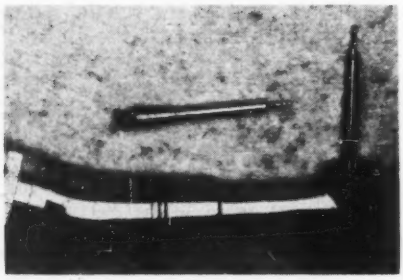

Fig. 1. Special drill in back-action handpiece. (Modified from the original design by Karlstrom[2]).

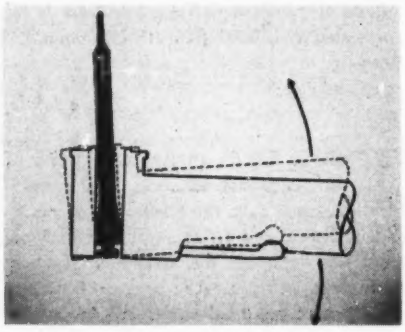

Fig. 2. Diagram of loose fit of drill in handpiece.

by this technique. Teeth which are overlapped present obstacles in designing the preparation. Subject to individual variations, the splint should be inserted in a line which bisects an angle formed by the lingual surfaces of the crowns of the incisors and the long axes of the incisor teeth. Consequently, teeth which are overlapped need to have sufficient reduction on the proximal angles to permit the splint to be seated in place.

Another more serious problem is the presence of large pulps in the teeth which are to be operated. Most of the time, however, teeth which are in need of this type of periodontal therapy present very small pulp cavities.

Because of stresses induced upon the gold castings of the splint, it is suggested that splints be limited to a span of 6 teeth or less, although 7 teeth have been splinted in a single unit with good results.

Design is begun by fitting the parallelometer on a study model and surveying the path of insertion and the location of the finish lines of the proposed castings. For the first few cases, it is suggested that the preparations be made on the study model. Actual preparation of the involved teeth and the impressions must be completed in one sitting. Usually 2 hours is sufficient for this first appointment. Tooth preparation is begun by using a diamond stone to reduce necessary enamel and dentin. Care must be taken to remove only a minimum of tooth structure on the lingual surfaces as this bulk of tooth structure will aid in the retention of the pins. Preparation of the proximal surfaces is also worthy of mention. Often undercuts are produced because the finish line may be carried too far labially and at the wrong angle. This is most likely to occur on the

Fig. 3. Jig mounted on dentiform.

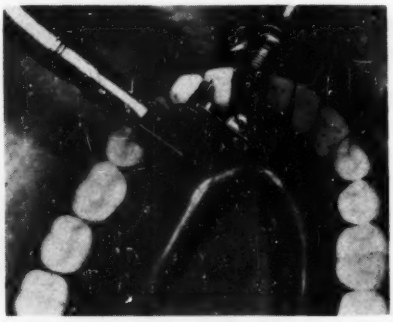

Fig. 4. Drilling of a pinhole on a dentiform tooth.

Fig. 5. Typical tooth preparations. Design on the right does not include the incisal edge.

Fig. 6. Nylon bristles in position for taking the impression. Note compound on labial to obstruct the gingival embrasures.

mesial surface of the cuspid. Margins must be clearly defined by a small chamfer so that the wax pattern can be accurately made. Moreover, the incisal edge, if it is involved, should be "hollow ground" to provide for adequate bulk of gold.

Following the diamond reduction and the discing of the cavity margins, the sites for drilling the pinholes are located and marked with a pencil. At least 3 pinholes should be used in each incisor and 4 to 6 pinholes in each cuspid. Following the marking of these pinholes with a pencil, a spearpoint carbide bur is used to "center" the hole for drilling. A No. ½ carbide bur may also be used. This is done to penetrate all remaining enamel and to give the drill a starting point.

Blocking off the saliva with cotton rolls and providing an absolutely dry field is done prior to mounting the aligning jig with compound. The field must be kept dry and the compound mount firm and rigid during the entire drilling operation, otherwise the jig will loosen and a loss in parallelism will result. Using the special drill in a back-action handpiece, the drilling is done with the motor revolving at slow speed. If the size of the pulp cavity permits, each pinhole is drilled to a maximum depth of 2½ mm. If there is a possibility of pulp exposure, a pinhole of at least 1.0 mm. in depth may be drilled directly toward the pulp without fear of penetration.

Following the removal of the aligning jig, the area is prepared for taking the impression. Mercaptan rubbers or silicones are the impression materials of choice.

To facilitate reproduction of the pinholes, special nylon bristles are used.[3] These bristles measure .022″ in diameter and approximately 5 mm. in length and have a knob on one end to engage the impression material. These are inserted in the pinholes as can be seen in Figure 6.

Conventional impression trays are unsatisfactory because of the lingual inclination of the pinholes. Because of this difference, it is impossible to obtain a reproduction of the labial surfaces of the tooth. A flat piece of tongue blade, or an old bur box, trimmed to fit the lingual curvature of the incisor teeth makes a suitable tray

Fig. 7. Impression removed from the teeth. Note the black nylon bristles protruding from the light colored silicone impression material.

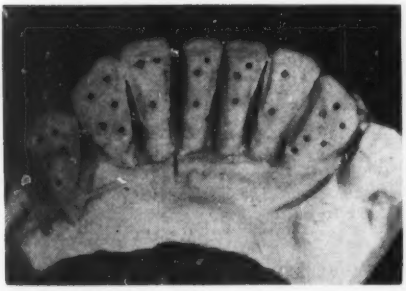

Fig. 8. Stone die which has been trimmed. Black spots indicate pinholes left by .022" nylon bristle which was used for taking the impression.

Fig. 10. Above: Teeth before splinting. Below: Splint in place.

for obtaining a reproduction of all margins and the lingual surfaces of the involved teeth. It is important, however, that the gingival embrasures of the teeth be well blocked off with compound or wax so as to prevent material from extruding labially through this space and locking the impression into position. Removal of such a "locked" impression would tear the material and induce distortion. After the impression has been poured up in die stone, the model is trimmed from the labial to clear and free all margins.

As can readily be understood, the fabrication of an accurate wax pattern to fit miniature pinholes is difficult. E. D. Shooshan[4] is responsible for devising a simplified technique of making a wax pattern using nylon bristles in conjunction with inlay wax. In the above case, using .022" bristle for the impression, .020" diameter bristle is used for fabricating the wax pattern. Nylon bristle is supplied from the manufacturer in 3 inch lengths.[3]

Fig. 9. Finished splint ready for cementation. Note length and multiplicity of pins.

In order to have the bristle fit loosely within the pinhole of the die, the end should be cut squarely with a sharp razor blade. After being inserted in the pinhole, a hot instrument can be used to cut the bristle, leaving 0.5 mm. extending from the hole. The heated instrument tends to leave a ragged edge which engages the inlay wax making a more rigid attachment for the bristles. Care must be taken to be sure the die has been previously well lubricated. Inlay wax is flowed over the bristles and a wax pattern is formed of each individual tooth. For ease of fabrication, two of these wax patterns are attached to a single sprue, invested and cast by means of centrifugal or vacuum machines. Routinely, for strength and rigidity, hard partial denture gold is used. The nylon bristle burns out of the mold and a clean, accurately fitting gold pin is left in its place.

The orifices of the pinholes in the teeth are chamfered with a No. 2 or 4 round bur to permit a more complete seating of the casting. After the castings are fitted on the individual teeth, a lingual key in plaster is taken of the castings in position and the assembly soldered into one complete splint.

CEMENTATION

Cementation is done with zinc phosphate cement. It might be expected that air would be trapped within the pinholes.

Fig. 11. Roentgenograph of case before splinting.

Because of the large number of pinholes present, it is easily seen that the operator is confronted with an impossible task to work cement into each of the pinholes prior to the placement of the casting. However, cementation is no problem if the following method is used:

Thirty or forty drops of cement liquid are placed upon a cool slab so as to provide a large mass of cement. It is mixed slowly to a thin inlay setting consistency. The lingual surfaces of the teeth are wiped with a copious supply of this cement; none need be placed upon the casting. The splint is then seated in place. Next the splint is withdrawn about 1½ mm. from its seating position, and another supply of cement is scraped under the incisal margin. The splint is then seated a second time. Again the splint is withdrawn a short distance, more cement introduced and the splint seated to its final permanent position. The gold pins act as tight-fitting plungers inside the walls of the dentin to drive out the air. When they, in turn, are withdrawn, the hydraulic pressure sucks the cement into the depths of the pinholes, insuring a homogeneous cement bond, free of all air bubbles.

Fig. 12. Preparation complete. Note pinholes and conservation of tooth structure.

DISCUSSION

This technique is not difficult nor time consuming. However, two precautions are worthy of mention. First, this technique is different from conventional procedures and many pitfalls may lie in the path of the inexperienced. Therefore, it is suggested that for the first case the complete operation be performed in extracted teeth which are aligned in an arch form comparable to that of the patient.

The second precaution pertains to the scheduling of patient appointments. Because of the necessity of keeping the pinholes free of foreign material, it is essential that they be sealed over with quick curing acrylic. Waxed sewing thread[3] is used in the pinholes to keep them free from acrylic while the castings are being fabricated. Because of the percolation of saliva around the acrylic, the pinholes are accessible to saliva, and a temporary should not be left in place more than two weeks. Indeed, because of overall problems associated with temporary protection of the cavity prepa-

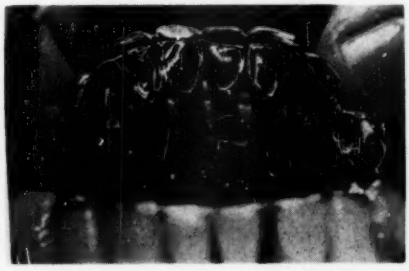

Fig. 13. Finished splint, lingual view.

rations, it is highly recommended that appointments be long and spaced as close together as possible.

The complete splinting operation is depicted in Figures 6 to 13. As can be seen from the radiograph (Fig. 11), the prognosis of these mandibular incisors would be virtually hopeless without splinting. With due regard for conservation of tooth structure and esthetics, this patient is now provided with a 28-pin, fixed splint, stabilizing 7 teeth. At the time of writing, the splint shown above has been in place for several months with excellent results.

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PHILADELPHIA SOCIETY OF PERIODONTOLOGY

The Philadelphia Society of Periodontology announces an all-day dental meeting on January 18, 1961. The meeting will be held in the VA Hospital, University and Woodland Ave. at 1 p.m. The subject will be Mucogingival Surgery; Dr. Emanuel H. Malamed will be Moderator of a panel consisting of Dr. D. Walter Cohen, Col. Harold Grupe, Dr. Frederic M. Chacker, Dr. Theodore West, and Dr. Harry Bohannan. Fee, Members, $20, Non-Members, $25 and Students $5.00.

An all-day medical meeting will be held in conjunction with the Philadelphia Chapter of the Academy of Dental Medicine on Wednesday, February 15, 1961. The subject will be "Diabetes—Its Relationship to Dentistry" and the panelists will include Joseph T. Beardwood, Jr., M.D.; Norman G. Schneeberg, M.D., Emanuel Cheraskin, D.M.D., M.D. Fee, Members, $20; Non-Members $25 and students $5.

For further information write Dr. E. H. Malamed, Secretary, Philadelphia Society of Periodontology, 1147 Magee Avenue, Philadelphia, Pennsylvania.

Ultrasound and the Treatment of Acute and Chronic Gingivitis

by SOL J. EWEN, D.D.S.,* *Great Neck, New York*

ULTRASONIC vibrations possess mechanical, thermal, and chemical properties for the alleviation of periodontal disease. The probability exists that these properties of ultrasonic vibrations may have direct applicability in the treatment of gingivitis. This gingival disorder consists of a necrotic bed of dental papillae and gingival margins within which a profusion of organisms is harbored. The gingiva is usually edematous and may bleed easily; the necrosis may extend beyond the bottom of the sulcus and may involve the epithelial attachment. In the chronic form of gingivitis the visible tissues are pink and fibrotic; yet minute ulcers, not visible to the naked eye, persist deep in the periodontal or gingival tissues.

To treat the disorder current therapy advocates early debridement of the necrotic tissue supplemented by the use of antibiotics, gingival lavage, improved diet, and rest. In order to cure acute and chronic forms of gingivitis, meticulous scaling and curettage are essential.

This paper presents a technique based wholly on the utilization of ultrasonic vibrations for treating gingivitis. With ultrasound it is possible to debride the tissues, to produce sulcular lavage, to suffuse local tissues with antiseptics, and, when necessary, to excise or scarify soft tissues to alter gingival architecture.

MATERIALS

A round orthodontic tube (.030 o.d., approximately three fourths of an inch in length) is brazed to a magneto-strictor with silver solder. A moderately flexible steel tube (.022 o.d.) is then slipped through the original tube. Once the smaller one is in position so that it protrudes eight millimeters beyond the .030 tube, it is spot-welded into position. After heat softening, a series of two to three turns is made at the mesial end of the smaller tube. Upon completion of the turns and the addition of a thick end-collar, the tubes together are heated and water-quenched to harden. The distal end is pinched flat so that it may enter gingival crevices and periodontal pockets with facility (Fig. 1). This tubular device may now be vibrated at 25 Kc. Such a vibrating tube, ultrasonically activated, can mechanically remove root surface accretions, flush out the contents of gingival pockets, and warm or heat surrounding tissue (Figs. 2 and 3).[1,2] This heat may be used for micromassage and circulatory

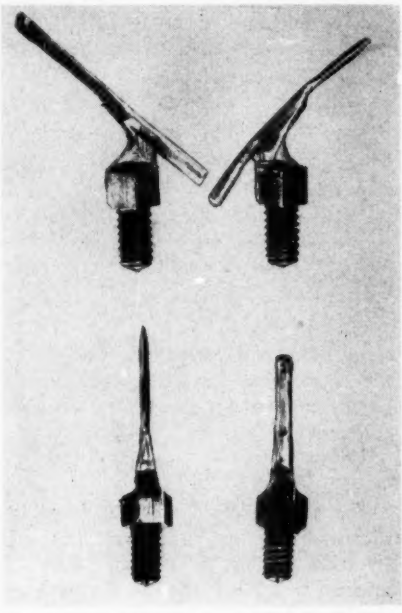

Fig. 1. Early design of periodontal tubes using orthodontic tubing.

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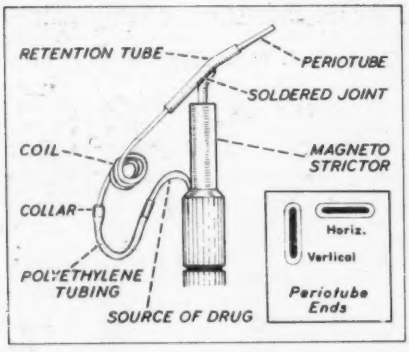

Fig. 2. Diagram showing construction of new perio-tube.

stimulation. If pressure is exerted by the tube against the tissue, it coagulates small bits of the surface, simulating soft tissue curettage.

METHOD OF TREATMENT

The tissues to be treated are painted with a disclosing solution; such as iodoglycerine, isodine, or gentian violet 2% aqueous, which is applied to the teeth and gingiva and permitted to soak in (Fig. 4). A fresh solution of sodium hypochlorite (one tablespoon of 2% Zonite diluted in a quart of tap water is mixed and placed into a pressurized tank (e.g. a garden tank for spraying trees) (Fig. 5). The antiseptic solution is maintained at a pressure of twelve to fourteen pounds. This antiseptic solution which may be sodium hypochlorite, hypertonic saline, or Ringer's solution with 10% magnesium sulphate is now directed through the ultrasonic generator and out into the handpiece. Proper fluid regulation permits the antiseptic solution to emerge from the vibrating tube at 39 degrees Centigrade, a temperature that is warm yet tolerable to the patient. The tube described above inserted into the handpiece produces during ultrasonic vibration a rapidly propelled fog or jet of antiseptic solution, about one and one half inches wide. This jet, confined between the gingiva and the tooth, flushes out all soft accumulations. Simultaneously, tiny white curds of gingival tissue which have been locally coagulated are also flushed out of the gingival crevice. Such gingival sonations or treatments may, under local anesthesia, be repeated every forty-eight hours, until full resolution of the inflammation has taken place.

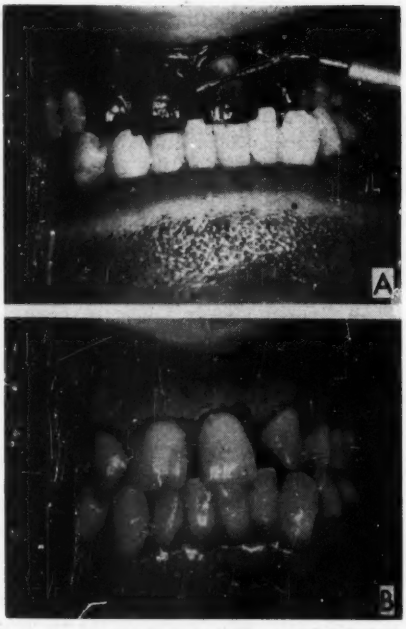

Fig. 4. A—Teeth flooded with gentian violet 2% aqueous. B—Dye bleached out sodium hypochlorite vibrated ultrasonically.

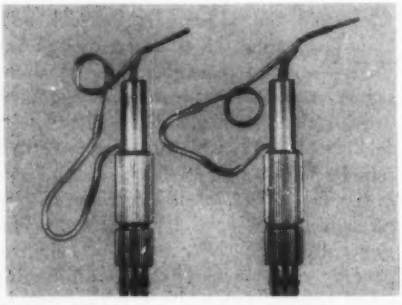

Fig. 3. Perio-tubes with vertical and horizontal tips.

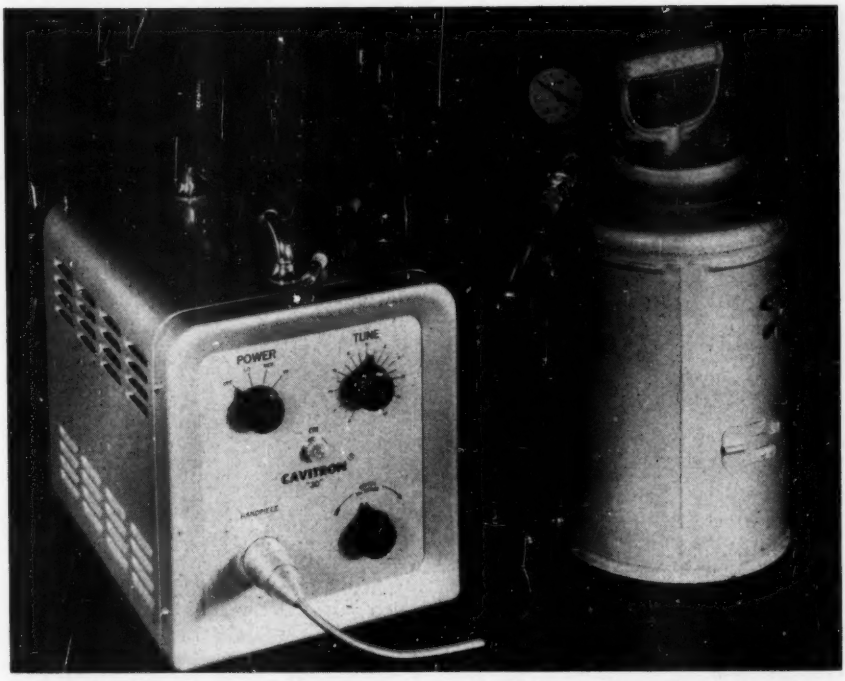

Fig. 5. Ultrasonic generator with pressure tank on the right containing antiseptic at twelve to fourteen pounds pressure.

CASE HISTORIES

1. M.E., male, age 26, presented with acutely painful edematous hemorrhagic gingivae of three weeks standing (Fig. 6). A 3% iodine solution was painted over the teeth and gingiva. Sodium hypochlorite solution in an ultrasonic spray was directed through a tubular ultrasonic tip. Once inflammation had subsided, bleeding disappeared, and the tissues returned to a firm pink state, although shiny and translucent in appearance. Gingivoplasty to correct the architecture was initiated. The treatment was completed in eight sessions.

2. F.L., female, age 50, presented with a recalcitrant form of chronic marginal gingivitis. Tissue was thickened and malformed, and the interdental spaces were filled with materia alba and food. Cervical areas of the teeth were stained and covered with plaque. The gingiva was sensitive to touch and in some areas bled easily on instrumentation. All previous treatment had been only partially satisfactory. Sodium hypochlorite was again used in treatment. Four sonations, several weeks apart, were sufficient to facilitate normal healing (Fig. 7).

DISCUSSION

The effects of ultrasonic vibrations on animal and human tissues have been studied extensively in the last fifteen years. In periodontics specific investigation of the effects of ultrasound on human gingiva have only recently begun. W. J. Fry,[3] in applying ultrasound through an opening in the calvarium to the central nervous system of an animal, has been able, using

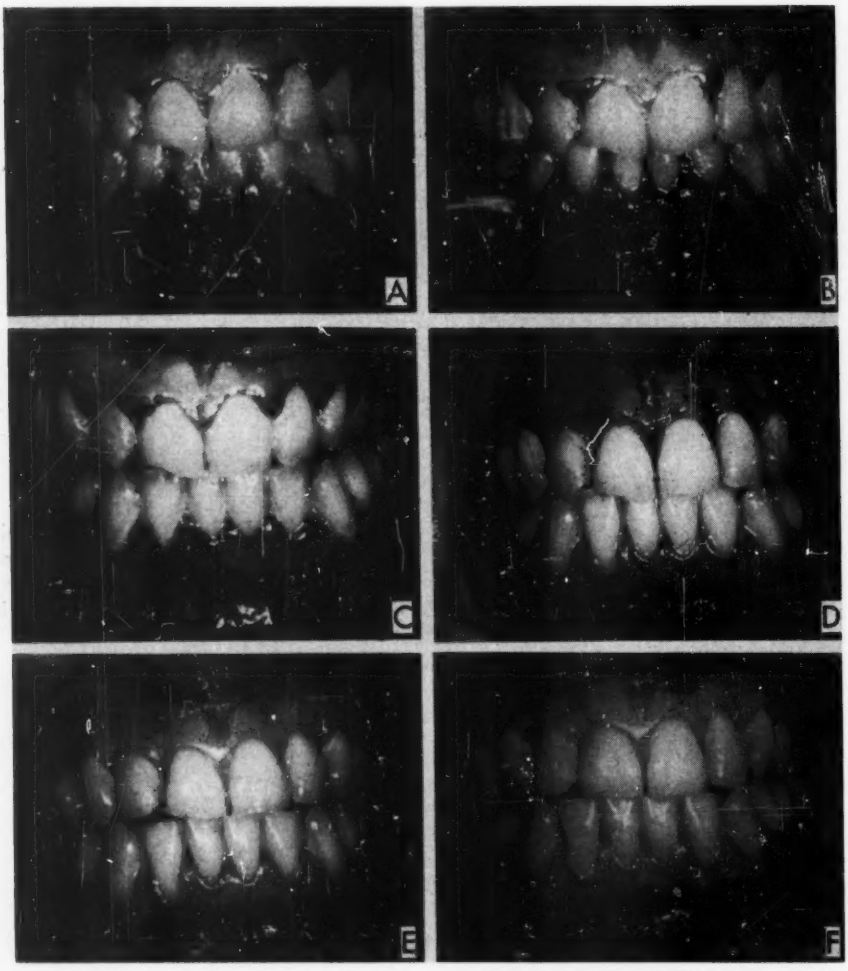

Fig. 6. Acute necrotizing ulcerative gingivitis. Ultrasonic treatment with sodium hypochlorite. A—Lower anterior teeth before treatment. B—Immediately following first treatment. C—One week. D—One month. E—Six weeks. F—Three months. (Eight sonations).

Ringer's solution as the conduction medium, to produce reversible lesions. Hesselberg,[4] through the use of cavitation or bubble-forming effects, has been able to stimulate cell division. Using ultrasound on BCG in suspension, he has shown a positive dispersing and bactericidal effect. Applying these findings to periodontal therapy, one may postulate that mild antiseptics vibrated at ultrasonic speed are able mechanically to debride necrotic material, perfuse the tissues for some time, and possibly destroy organisms at the local site. It has been shown clinically that gingival wounds created in ultrasonic curettage heal without untoward sequelae.[5]

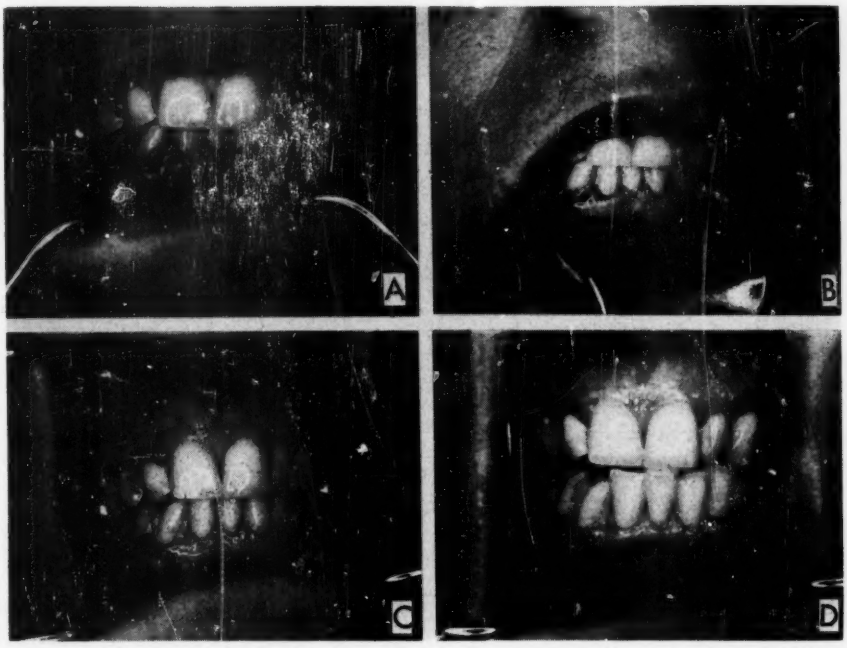

Fig. 7. Chronic marginal gingivitis. A—Lower anterior teeth before treatment. B—One week. C—One month. D—Three months. (Four sonations).

SUMMARY

A steel tube which vibrates at ultrasonic frequencies has been constructed. Utilizing a closed pressurized system of drug dispersion, antiseptics have been directed at inflamed human gingiva. Two cases where ultrasonic drug dispersion treatment has been used in periodontal therapy have been presented.

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Gingivitis in Children of Israel

by KURT A. ROSENZWEIG, D.M.D., M.P.H., *Israel*

ALTHOUGH the epidemiology of dental caries has been investigated in a number of countries, the prevalence of periodontal diseases has received relatively little attention primarily because a standardized index for the quantitative assessment of periodontal disease is not yet available. The P-M-A index for the assessment of gingivitis in children was proposed by Schour and Massler in 1949.[28] Russell[19] has suggested a method of estimating periodontal disease in adults. Both methods have been used to a limited extent by a few investigators.

This study presents the average P-M-A in a sample of 4,500 children, 13-14 years of age, living in Israel. These children represented seven different ethnic groups living under different environmental, dietary and cultural conditions. The findings in each group are therefore presented separately and compared.

MATERIAL

The study is based upon the examination of 4,500 children 13 to 14 years of age. The children were classified in seven different "strata" according to the father's country of birth. *"Near East"* included Jews from Syria, Lebanon and Egypt; *"North Africa"* included Jews from Libya, Algier, Tunis and Morocco. All European countries as well as both Americas were comprised in the stratum of *"Europe."* *"Iraq and Iran"* as well as *"Yemen and Aden"* were additional subgroups. *"Arabs"* were non-Jewish (local ethnic minorities). The group called "Israel" was composed of children whose fathers were already born in Israel. Similar ethnic and cultural patterns were a prerequisite for inclusion in a particular stratum. The design of the sample, some anthropologic background material as well as the statistical methods applied have been previously reported in a study of dental caries in these groups.[25]

METHOD

The examinations were carried out by two dentists who had been previously trained in the P-M-A method in order to make their findings comparable. Gingival inflammation was expressed in terms of the P-M-A index,[16] counted for the anterior segments only, although it has been argued that hereby a considerable amount of inflammation would thus be omitted.[2] The number of papillary (P), marginal (M) and attached (A) gingival units affected are presented separately. In addition, P was weighted by 1, M by 2 and A by 3, and then added together. It was felt that this might give an overall picture of the total inflammation present, since it is generally accepted that in most cases inflammation begins at the papilla, progressing via the margin to the attached tissues.[7] Besides it was thought that it would thus be possible to express of degree of severity of those "misfit" cases which would not coincide with the class intervals of severity selected in the Chicago study.[7] This modification of the index was introduced with the approval of Dr. Schour.

FINDINGS

Gingivitis was present in 43.11 to 82.62 per cent of the children, according to their origin (table 1). Children born in Israel showed the least amount of gingivitis while the North African Jews and the Arabs showed the highest (table 1). The average number of papillary and marginal areas affected were not statistically different in the groups from Israel, Iraq, Yemen, Europe and the Near East. However, the differences between them and North African group, and between the

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TABLE I

Prevalence of Gingivitis in Children 13–14 Years of Age by Origin. (Average P+M+A)

Origin	No. Examined	P % Aff.	P Aver.	M % Aff.	M Aver.	A	Weighted PMA	Males	Females
Israel	214	43.11	1.95	29.47	0.96	0.000	3.87	4.21	3.02
Yemen	369	52.99	1.96	37.63	1.27	0.003	4.51	5.16	4.13
Iraq-Iran	561	52.07	2.31	35.72	1.63	0.024	5.63	8.25	4.82
Europe	1946	52.43	2.45	37.77	1.73	0.014	5.95	8.27	4.00
Near East	156	55.39	2.71	36.26	1.78	0.000	6.27	6.75	3.65
North Africa	565	66.36	3.75	52.96	3.02	0.036	9.91	12.20	8.95
Arabs	587	81.92	6.22	82.62	6.11	0.017	18.50	19.25	16.35
Philadelphia	2,922	55%	2.7		1.2	0.0	5.1	5.4	3.5
Chicago children	1,929	61%	3.1		2.3	0.0	7.7	8.3	5.1

Recalculated for anterior segments of 13 to 14 year old children from data presented by Massler, Cohen and Schour (1952).

There were no statistical differences among the groups Israel, Yemen, Iraq-Iran, Europe and Near East in any P—M—A category. There were highly significant differences between all members of this group and North Africans ($P=0.01$ to 0.05) and between all groups and Arabs ($p=0.01$).

latter and the Arabs were statistically highly significant (table 1). The same relation existed between the percentages of children exhibiting gingivitis of any type and to any degree. The relation of marginal to papillary inflammation was remarkably constant in all groups except the Arabs, who not only had absolutely more gingivitis of both types than all other communities, but also relatively more marginal gingivitis (table 1). The North Africans exhibited the same type of gingivitis as did the other Jewish groups only to a higher degree, whereas that of the Arabs seems to be of a more severe kind. Original data available in detail, upon request.

In all groups gingivitis was more prevalent in males than in females (table 1). The differences were statistically significant in all groups except the Yemenites. These findings are in accordance with other investigations.[7,11] Various explanations have been offered, among them that girls generally pay more attention to oral hygiene and home care.[2] While this may be true for other groups it is not applicable to Arabs and other oriental communities in Israel who, to a great extent do not practice oral hygiene at all. It may be that these sex differences in gingivitis are due to hormonal factors operating at 13-14 years of age.

No significant differences were observed in gingivitis between children living in cities or villages. However there was a significantly greater amount of gingivitis in children living in Kibbutzim (settlements).

A large number of children examined were born in other countries and immigrated to Israel only after they had spent their early years elsewhere. A comparison was therefore made between native born children and immigrant children who had lived at least the first 6 years of their lives in other countries. In general, the prevalence of gingivitis was higher in the immigrant children (weighted PMA $= 7.26$ against 5.38). This was also true when city, village and Kibbutz children were considered separately.

DISCUSSION

A comparison of these data on gingivitis with the data on the distribution of dental caries in the same sample (14) shows that the two oriental groups, North Africans and Arabs, had the lowest caries experience and the highest gingivitis. The other groups showed no correlation either positive or negative between these two *diseases*.

A comparison of these data with those presented by Massler, Cohen and Schour on 13-14 year old children living in Phila-

delphia and in Chicago (table 1) shows a fairly good agreement between the two studies. The Israel group shows a slightly lesser amount of gingivitis than the American children while the North Africans and Arabs show a considerably greater amount of gingivitis.

It is customary to differentiate between systemic and local factors in the etiology of periodontal disease. For gingivitis endocrinologic disturbances, malnutrition and even racial or geographic causes have been made responsible.[6] The latter may be dismissed at once as explaining the differences found in the various population groups included in the present survey. Of the nutrients essential for the maintenance of gingival health ascorbic acid has been found most important. There is no doubt that fresh fruit and vegetables are abundantly present in the diets of the whole population of Israel during the whole year.[13, 19, 20] In the nutritional investigations carried out from 1951 to 1953 lower hemoglobin values were found in all groups investigated than in comparable reports from elsewhere.[19] It was felt that this might be due to an unsufficient intake of essential proteins rather than to lack of iron. Gingivitis and other oral manifestations of malnutrition were observed only in the lower socioeconomic strata and attributed to deficiency in vitamins of the B group. On the whole there is not much recent information available on the nutritional status of the different population groups in Israel, and so dietary factor can at present account for the differences encountered in gingival health. Changes in the gingiva of boys and girls during the pubertal period have been reported, and the hormonal state has been pointed out as the predisposing factor.[4] There is, however, not much knowledge up to now exactly how hormonal dysfunction occurring frequently during puberty manifests itself in the periodontal tissues.[1] Nothing is known at all so far whether there exist differences in the onset of puberty among the population groups in Israel, therefore the influence of hormonal factors on the gingival status cannot be appraised.

Of local factors faulty dentistry, faulty hygiene, defects of the occlusion, habits and calculus have been mentioned.[3] The children examined in the present survey did not receive much dental care,[14] thus the first factor cannot be of importance. No attempt has been made to assess the quality of mouth hygiene on the ground of debris, materia alba etc. found at the examination as was done elsewhere.[2] It was felt that this is not a very reliable indicator, and besides easily influenced by examiners' bias. However, it has been shown in anthropologic surveys[13, 18] that oral hygiene is largely unknown among the Arabs in Israel, as well as among a vast majority of the immigrants from oriental countries. This fact may at least partly account for the high prevalence of gingivitis among Arabs and North Africans. It appears that in this regard the immigrants are favorably influenced the longer they live in Israel. An intensive, continuous campaign in health education including oral hygiene is carried out in hospitals, clinics, maternal health centres and schools, and is certainly responsible to some extent for the good gingival conditions prevailing among the "Israel" group. It has been shown that the most adaptable and progressive community among the oriental Jews are Yemenites,[6] and this fact is reflected by their gingival health. The rising standard of living after immigration plays its part not only in raising personal hygiene, but also in an improved nutrition. Thus both general and local factors work together, the former furnishing the background, and the latter being the trigger in the etiology of periodontal disease.[5] It appears that a variety of factors, partly interdependent causes are accountable for the fact, that there is more gingivitis in new immigrants than in the old established population in all forms of settlement with the notable exception of the Kibbutzim.

Some importance in the etiology of gingivitis has been attributed to the physical character of the diet whose detergent

properties are said to exert a stimulating and cleaning influence on the gingiva, but "a survey of the available literature indicates that there have been no convincing studies reported to show that the physical character of the diet bears a proved relationship to the prevention of periodontal disease."[1] Besides, nothing is known about the physical properties of the diets eaten by the various population groups in Israel.

GINGIVITIS AND MALOCCLUSION

Children in Kibbutzim are much more afflicted by malocclusion than either in town or in village, and among them especially those born or raised in the Kibbutz (collective settlement). It is possible that this circumstance is responsible for the high prevalence of gingivitis in Kibbutz-children in general, and in the locally reared in particular. This assumption was supported by the fact that the correlation coefficient between gingivitis and malocclusion, calculated for all Jewish children, in the sample was positively significant ($r = 0.65$). Gingivitis was more prevalent among children of the old established residents and or immigrants in villages than among urban children. This finding is understandable considering that: The percentage of immigrants of oriental origin is much higher in villages than in the cities; dental care is much less available; the socioeconomic level is on the whole lower. No statistically significant difference was found between Arab children in town and village, but here too a significant correlation between gingivitis and malocclusion was established ($r = 0.57$). No attempt was made to group gingivitis according to severity, as done by Massler and Savara,[15] who found an association between both diseases only when gingivitis was of the severe type.

SUMMARY

The prevalence of gingivitis was assessed in 4,500 children, 13-14 years of age, living in Israel. The children were drawn from seven different ethnic groups (table 1). Two dentists who had been previously trained and calibrated for comparability carried out the investigation by means of mouth mirror and explorer. Gingivitis was registered for the anterior segments only in terms of the P-M-A index. In order to express the total amount of inflammation, the components of this index were weighted by $P = 1$, $M = 2$ and $A = 3$ and then summed up. Averages were calculated for subgroups according to sex, origin, sojourn in Israel and type of settlement.

CONCLUSIONS

In general the prevalence of gingivitis in 5 ethnic groups living in Israel was very similar to that reported for children living in Chicago and Philadelphia. Gingivitis was significantly higher in North African Jews and very high in Arabs (table 1). The Arabs showed also comparatively more marginal inflammation, the ratio between papillary and marginal gingivitis being nearly constant for all other groups (table 1). Oral hygiene, food habits, nutritional status and malocclusion were the etiological factors considered. A positive correlation between gingivitis and malocclusion was established in both Jews and Arabs. Boys had more gingivitis than girls at age 13-14 years.

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A Study on the Relationship Between Bruxism and Aggression*

by JOE FRISCH, D.D.S., MAJOR, DC, USA,** LAWRENCE KATZ, PH.D.,† AND ANTONIO J. FERREIRA, M.D.‡

INTRODUCTION

WHILE much has been written on the etiology of bruxism, it is still a topic which is far from well understood. The relationship of bruxism to periodontal disease, temporal mandibular joint disturbance and neuropsychiatric conditions requires further clarification. Orban and co-editors[1] define bruxism as grinding, gritting or clamping the teeth. It may occur consciously or unconsciously during the day, or unconsciously during the night. This definition has been stated previously by others.[2,3,4,5]

Concerning the etiology of this condition, many authors have considered bruxism to be directly due to the oral expression of aggression. Miller writes: "In every case of grinding of the teeth there is a defect in the measures which the personality employs to inhibit aggression. In every case of bruxism the core of the problem is to be found in the functioning of the personality."[2] Goldman and co-authors[6] state: "Psychiatrists regard bruxism and clenching as the oral outlet for subconscious aggressions." The role of aggression, tension, and other emotional factors as causative of bruxism is postulated in many publications.[7,8,9,10,11,12,13,14] An excellent review of the literature can be found in Nadler's publication.[10]

Although a direct relationship between patterns of expressing aggression and bruxism has repeatedly been hypothesized, research to test this hypothesis has been minimal. It was for this reason that the authors undertook the present study. (The interesting question of the possible relationship between clenching and aggression, while in the same general area as the topic under investigation, is not specifically studied in the research.) If it is true that bruxism is the result of the oral expression of aggression, then it might be expected that individuals with evidence of a high degree of bruxism would demonstrate either a greater tendency to respond to frustrating situations with aggression or a greater tendency to turn aggression inward than patients with less bruxism. In somewhat more operational terms, the hypothesis underlying the present study may be stated as follows:

Patients with evidence of a high degree of bruxism demonstrate different patterns of expressing aggression on a psychological test than patients with low or moderate amounts of bruxism.

METHOD

The study was executed in the Periodontia Section of the Dental Service, Letterman General Hospital, San Francisco, California. Participants in the study were 95 patients who were referred for evaluation and treatment and had had no prior treatment in this section.

While awaiting his initial appointment, the patient was greeted by the dental nurse and asked if he would be willing to participate in a research program. If a patient agreed to participate, he was requested to fill out a very brief questionnaire concerning basic identifying data, was asked to draw a picture of a person, and was administered the Rosenzweig Picture-Frustration

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Study. (This instrument, described below, was the main focus of interest in the present study.) An area was set aside for the subject to work, providing privacy and quiet. If any hesitancy was shown by a patient, the matter was dropped and treatment was given as required; however, there were not many refusals. After completing the various materials of the study, each patient placed them in an envelope, which he turned in to the nurse, and then was seen for his regular appointment.

Alginate impressions of the patient's dentition were taken and poured in stone. Natural dentition having a minimum of 16 teeth was required. These models were used to determine the degree of non-functional abrasion. An additional direct examination of the patient's dentition to determine the evidence of abrasion was accomplished and recorded. The amount of non-functional abrasion was used as the measure of bruxism. To insure consistency, rating of all study casts was performed at one sitting, after all subjects of the study had been seen and without reference to ratings based upon direct examination. Bruxism was rated on a three-point scale as minimal (Class I), moderate (Class II), or excessive (Class III). For greater refinement, plus and minus ratings were also made in each of the three categories. Using the above definition of bruxism, two different dentists independently rated the degree of bruxism indicated on casts, on the first fifty patients in this study. In more than 90 per cent of these cases, disagreement was less than one category apart and generally the scoring coincided. Almost all subjects demonstrated some degree of bruxism with ratings of "moderate" being most frequent. It was a common occurrence to find flattening of the occlusal anatomy, as well as faceting localized to a limited area rather than generalized throughout the dentition.

The instrument used to assess the subject's characteristic modes of expressing aggression was the Rosenzweig Picture-Frustration Study.[15] This instrument is a booklet containing 24 cartoon drawings, each depicting some frustrating situation. Subjects are told to write in the replies which the frustrated person might make in each of these situations. Each of these replies is scored, according to a system outlined in the scoring manual,[16] and several measures may be obtained from these scores. Of greatest relevance to the present study are the measures of direction of aggression shown in response to frustration, and the extent to which any kind of aggression is shown. A response may be scored as "extrapunitive," "intropunitive," or "impunitive." An "extrapunitive" response is defined by Rosenzweig as a response in which aggression is expressed and directed against the environment; an "intropunitive" response is one where aggression is expressed but turned by the subject against himself; and an "impunitive" response is defined as one where no aggression is expressed in response to a frustrating situation. Measures of the degree to which each of these three modes of expression is verbalized may be obtained by following scoring instructions in the manual.

RESULTS

Before comparing ratings of the degree of bruxism with measures of aggression, an attempt was made to assess the reliability of each of these measures. Ratings of the degree of bruxism made by direct observation of the subject's dentition were compared with ratings based upon cast impressions. Very close agreement was obtained, subjects almost always falling in the same category (Class I, II, or III) by each of these two methods. In addition, ratings by two independent judges described above, showed very close agreement. Two different scorers, each of whom tried to follow the manual as closely as possible, independently scored the responses on the Rosenzweig Study of the same 26 cases. Again, agreement was very close, both raters agreeing within a few percentage points on all cases. Scores for the total group of 95 subjects used in later calculations were those of a single scorer.

TABLE I

Percentages of Subjects Showing High Versus Low "Extrapunitiveness," "Intropunitiveness," or "Impunitiveness," For Three Degrees of Bruxism

Bruxism	*Intropunitiveness*		*Extrapunitiveness*		*Impunitiveness*	
	% Low	*% High*	*% Low*	*% High*	*% Low*	*% High*
I. Minimal	58	42	48	52	63	37
II. Moderate	45	55	52	48	47	53
III. Marked	50	50	55	45	50	50

After the reliability of each of the measures had been established, measures of bruxism were compared with measures of aggression, to provide data bearing on the hypothesis stated above. Comparisons were made separately between the degree of evidence of bruxism shown on cast impressions and each of the three measures of mode of expressing aggression; extrapunitiveness, intropunitiveness, and impunitiveness. The results are summarized in Table I, which gives the percentages of subjects scoring either "high" or "low" on each of the three measures of aggression, at each of the three degrees of bruxism. A Chi-Square test of statistical significance[17] applied to these data indicated that none of the differences appearing in this table was statistically significant. Subjects whose casts indicated marked evidence of bruxism did not differ from those who showed moderate or minimal evidence of bruxism in the degree to which they demonstrated extrapunitiveness, intropunitiveness, or impunitiveness. As far as these results go, subjects with marked bruxism did not show more aggression turned toward the environment, toward himself, or both, than subjects with lesser degrees of bruxism.

DISCUSSION

The results of the present study do not support the hypothesis that bruxism is associated with aggression. Either this hypothesis is false, or else some defect was present in this study which prevented the demonstration of a relationship which in fact exists. The major possible sources of defect in the study center around the measurement of bruxism and the measurement of aggression. Concerning the measurement of bruxism, Sicher[18] and Gottlieb, Orban and Diamond,[19] point out that teeth wear through life as a result of their functional use, and it might be expected that functional wear patterns of chewing and swallowing might be difficult to differentiate from bruxism. The authors feel, however, that this differentiation can be made quite reliably and feel that the agreement of two independent judges reported above illustrate this point. The fact that ratings of bruxism made directly on the patient correlated very highly with ratings based upon cast impressions would tend to support this view.

There is somewhat more question about the measurement of aggression. Although the Rosenzweig Picture-Frustration Study has been found to correlate with behavioral indications of aggression in past studies,[15] this instrument has not been fully validated. "Aggression" has many different meanings to different authors and may refer to overt behavior, verbalizations, personality predispositions, or even unconscious processes. It is, therefore, difficult to measure in a way which would satisfy various theoretical formulations. Perhaps more to the point of the present study, the instrument used measured contemporary feelings and tendencies, whereas the bruxism observed in the subjects might have occurred at any time in the past. It was not possible to determine whether the subjects with evidence of marked bruxism were still active bruxators, and it is possible that the non-functional wear on their

teeth occurred at a time when they were expressing considerable aggression. Another possibility is that people who demonstrate evidence of marked bruxism do not express more or less aggression from other people, but use the mouth as their prime outlet for aggression to a greater extent than other people. If this is true, then they might not show scores on the Rosenzweig which would be different from people with little bruxism but might demonstrate a greater tendency to express aggression orally. To test this hypothesis an independent means of measuring a tendency to express aggression orally would have to be found. This would pose problems, but perhaps some technique employing electromyography or electronic stress gauges might be feasible.

Because of these potential sources of error, the authors feel that there still remains a good possibility that bruxism and aggression are in some way related. On the other hand, there is no direct evidence in the literature which would clearly support this hypothesis. The authors hope to further explore this relationship in future studies, and hope that other writers, who feel that such a relationship exists, will attempt to demonstrate it through research on the subject.

ACKNOWLEDGMENTS

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Effects of Stress on the Periodontal Tissues of Young Adult Male Rats and Hamsters

by O. P. GUPTA, B.D.S., M.S., M.S.D., DR. P.H.,* HARRY BLECHMAN, D.D.S., M.A.** AND S. S. STAHL, D.D.S., M.S.***

A VARIETY of stressing agents have been used in an attempt to study their effects on the periodontal tissues of experimental animals.[1-8] Fedi[7] produced stress in young hamsters by successive exposure to cold, water bath and violent exercise, and a subcutaneous injection of 10 percent formaldehyde. He noted minor changes in the periodontal membrane fibers and fibroblasts. Shklar and Glickman,[8] using subcutaneous injections of 4 percent formaldehyde as a stressor in rats did not find any notable changes in the periodontal tissues of the stressed animals. This experiment, however, was of short duration and designed primarily to produce the alarm reaction.

The purpose of the present study was to investigate the response of the periodontium to long term stress produced by altering the environment of young adult male rats and hamsters.

EXPERIMENTAL PROCEDURES

Two experiments were conducted on two different animal species, rats and hamsters. The first experiment utilized 40 white rats weighing an average of 100 grams at the beginning of the experiment. Twenty-five rats were exposed to stress; the remaining 15 were kept as untreated controls. In the second experiment, 50 Syrian hamsters weighing an average of 75 grams were used. Thirty were stressed while 20 were kept as controls.

The animals of the experimental groups were subjected to stress which consisted of irritation and interference with their sleep by ringing of bells and exposure to bright lights. A cyclic pattern was followed. During the first 24-hour phase, bells rang for 90 seconds every 10 minutes. During the following 24 hours, the animals were exposed continually to bright lights from six 250 watt bulbs placed at a distance of 8 feet from the cages. During the next 24 hours they were not stressed. This cycle was carried on for 12 consecutive weeks.

All animals were housed in individual wire bottom cages. The control animals were housed in separate quarters. Ground Purina chow was provided *ad libitum* for a period of 12 weeks, after which the animals were sacrificed. The gross periodontal lesions in the soft and calcified tissues were evaluated by the method of Gupta and Shaw.[9] Representative mandibles were dissected out and prepared for histologic study. The tissues were fixed in formalin, sectioned at 7 micra, and were stained with hematoxylin-eosin.

Gross observations:

A large number of deaths occurred in the experimental groups, probably due to the extreme stress. The stressed animals grew at a much slower rate and attained lower final body weights than the controls. The average body weight at the end of the experiment for the control rats was 190 $\pm$ 8.2 grams and for the stressed rats 105 $\pm$ 15.5 grams. Similarly, the average final body weight for the control hamsters was 122 $\pm$ 7.3 and for the stressed hamsters was 74 $\pm$ 12.9 grams.

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TABLE I

Effects of Stress on Incidence of Periodontal Lesions in Experimental Animals*

Number of Animals at Beginning	Number of Animals at End	Duration (weeks)	SOFT TISSUES: Number of Periodontal Lesions Av.	SOFT TISSUES: Number of Periodontal Lesions C.R.**	SOFT TISSUES: Extent of Periodontal Lesions Av.	SOFT TISSUES: Extent of Periodontal Lesions C.R.**	CALCIFIED TISSUES: Number of Periodontal Lesions Av.	CALCIFIED TISSUES: Number of Periodontal Lesions C.R.**	CALCIFIED TISSUES: Extent of Periodontal Lesions Av.	CALCIFIED TISSUES: Extent of Periodontal Lesions C.R.**
EXPERIMENT 1. RATS										
Control 15	14	12	7.8 (0.5)	0.7	8.1 (0.5)	0.9	4.1 (0.4)	0.7	6.1 (0.4)	1.3
Stressed 20	12	12	8.2 (0.4)		8.9 (0.7)		4.6 (0.6)		7.2 (0.8)	
EXPERIMENT 2. HAMSTERS										
Control 20	16	12	22.5 (0.8)	1.7	29.6 (1.0)	0.7	16.9 (1.2)	2.7	19.0 (1.5)	2.5
Stressed 30	13	12	24.4 (0.7)		30.9 (1.6)		20.9 (0.9)		25.1 (1.9)	

*The values in parentheses are the standard error of the mean.

**C.R.—Critical ratio is the ratio of the difference between two means to the standard error of the difference between the means. Wherever the critical ratio is less than 2.0, the difference between the means is considered to be statistically insignificant; when the critical ratio is between 2.0 and 2.9, the difference is of borderline significance; when the ratio is 3.0 or higher, the difference is highly significant.

Fig. 1. Photomicrograph of an interproximal area between the first and second mandibular molars in a control hamster. Hematoxylin-eosin stain. Magn. 50X. Note the food impaction and inflammation in the gingival papilla.

The results of the gross evaluation of periodontal lesions of the animals of both experiments are given in Table I. The data in Table I indicate that the stressed rats developed somewhat more numerous and extensive soft and calcified tissue lesions than did the controls. However, the differences were not statistically significant.

The stressed hamsters developed more numerous and extensive soft and calcified tissue lesions than their controls. The differences in the number and extent of soft tissue lesions when compared to those of their controls were not statistically significant. However, the differences in the number and extent of calcified tissue lesions between the control and stressed hamsters were of moderate statistical significance.

The gross findings thus indicate that periodontal lesions did not increase sufficiently in rats to yield significant results, whereas they did in the hamsters, at least in the calcified tissues. It appears that under the experimental conditions stress was not associated with gingival soft tissue lesions but was associated with slight alveolar bone changes.

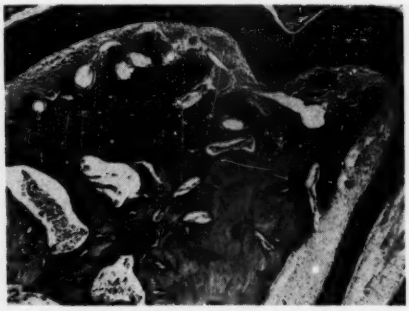

Fig. 2. Photomicrograph of the interradicular area of the specimen shown in Figure 1. Hematoxylin-eosin stain. Magn. 100X. Note the normal appearance of the alveolar bone.

Histologic Observations:

Since the histologic changes in the periodontal tissues of rats and hamsters were found to be identical, the changes observed will not be described by species.

Control Animals: Common changes observed in these animals were the food impaction areas in the interdental papillae associated with local inflammation. The epithelial attachment was at the cementoenamel junction. The alveolar crest in all animals showed slight remodeling with some evidence of osteogenic activity. The interradicular septum was normal. (Figures 1 and 2)

Stressed Animals: Slight to moderate impactions of food were observed in the interdental papillae (Figure 3), which were associated with local inflammatory infiltrations. The epithelial attachment was at the normal level. In some animals the periodontal membrane fibers were disoriented and were edematous. The most pronounced changes were observed in the alveolar bone. The alveolar crest showed areas of osteoclastic activity. A reduction in remodeling activity was noted. Marked and extensive endosteal osteoporosis was consistently seen

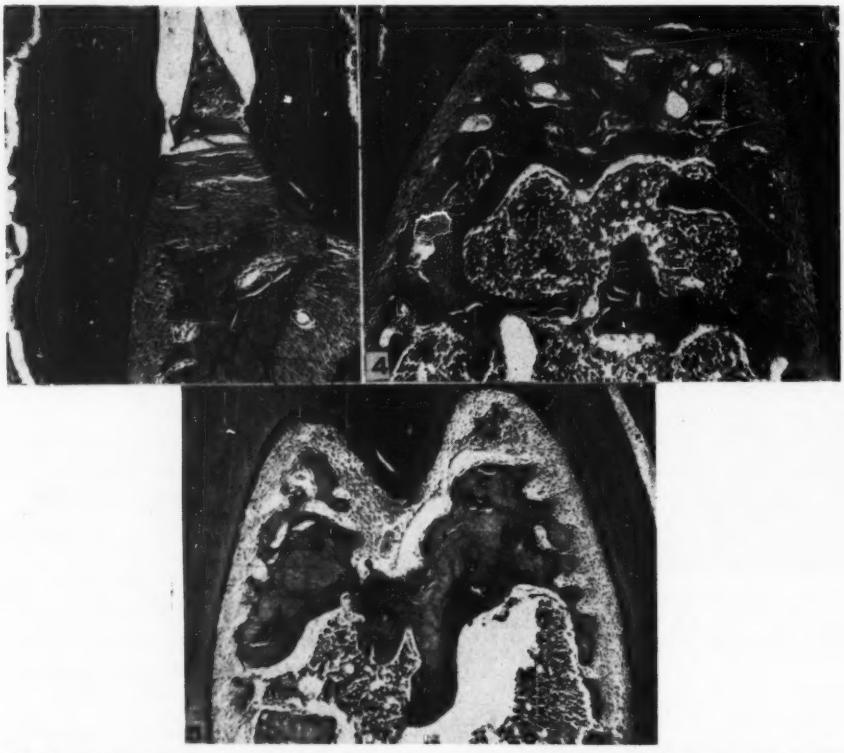

Fig. 3. Photomicrograph of an interproximal area between the first and second mandibular molars in a stressed hamster. Hematoxylin-eosin stain. Magn. 50X. Note the food impactions associated with local inflammation in the gingival papilla.

Fig. 4. Photomicrograph of the interradicular area of the specimen shown in Figure 3. Hematoxylin-eosin stain. Magn. 100X. Note the extensive endosteal osteoporosis in the interradicular septum.

Fig. 5. Photomicrograph of an interradicular area of a second mandibular molar in a stressed hamster. Hematoxylin-eosin stain. Magn. 100X. Note the extensive endosteal osteoporosis in the interradicular septum.

in the interradicular septi in all stressed animals. (Figures 4 and 5).

COMMENTS

The results of the above experiments indicate that relatively little change was produced in the gingival tissues of the rats and hamsters by long term exposure to excessive sound and light. However, the alveolar bone was consistently and extensively rarefied in all stressed animals. It is interesting to note that the two species differ in their susceptibility to lesions in their soft and calcified periodontal tissues upon the application of an identical stressor. It may be necessary in future studies to select species even more highly susceptible to periodontal disease than the hamster, as for example the rice rat,[10] in order to obtain significant increases in periodontal lesions under stress.

The data reemphasizes what has often been stated, namely, that soft tissue lesions depend primarily upon the presence of local irritants, whereas deterioration of calcified periodontal tissues is attributable to a major extent to systemic factors. These latter are of course potentially influenced by stress.

The endosteal osteoporosis produced in

the stressed animals is comparable to that reported in starvation,[11] protein deprivation,[1-6] and in tumor-bearing animals.[12] Since the stressed animals lost considerable weight during the experimental period, it is possible that the osteoporotic changes produced may be partially due to inadequate food intake. Further studies are necessary to define the exact role of stress in the etiology of alveolar bone changes.

SUMMARY

A study of the effects of stress on the periodontal tissues of young adult male rats and hamsters is reported.

Gross observations did not reveal significant differences in the soft and calcified tissue lesions between the control and stressed rats. However, stressed hamsters had significantly more calcified tissue lesions than those of the control group.

Histologically, extensive endosteal osteoporosis of the alveolar bone was found in the stressed rats and hamsters.

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Edward Bartlett Spalding, D.D.S.

September 18, 1868—November 2, 1960

Dr. "Ed." Spalding, widely known and respected member of the American Academy of Periodontology, died on November 2, 1960 in his home in Orchard Lake, Michigan. Dr. Spalding who had no peer in his knowledge or interest in the development of science of periodontology, was born in Eagle Harbor, Michigan and practiced in the Detroit and Birmingham area for 72 years.

In 1905 he became associated with Dr. Grace Rogers and the two worked diligently to develop and popularize the field of "preventive dentistry." The two doctors were married in 1910 and had long and fruitful careers together.

Dr. Spalding's many friends will miss his authoritative discussions on the progress that is being made in periodontology, his clever stories and his most unusual letters and Christmas cards. He was truly one of the early exponents of periodontolgy and was most emphatic about its importance. He was a past President of the American Academy of Periodontology as well as the Michigan State Dental Association. He was also a life member of the Detroit Boat Club, Omicron Kappa Upsilon, and Delta Sigma Delta.

Announcements . . . .

UNIVERSITY OF PENNSYLVANIA

Research and Teaching Traineeships in the basic sciences, in periodontology and in the comprehensive treatment and rehabilitation of the cleft palate patient are available at the School of Dentistry, University of Pennsylvania.

Candidates for the traineeships in the basic sciences must qualify for admission to the Graduate School of Arts and Sciences. These traineeships may lead to M.S. or Ph.D. degrees.

The traineeship in periodontology is a 2 year program devoted to basic science studies, clinical practice and research. It is intended primarily for those individuals who wish to enter into teaching and research in periodontology.

All traineeships are supported by the U. S. Public Health Service. Annual stipends vary from $4,500 and up depending on the qualifications and needs of the successful candidates. Inquiries for the basic science and cleft palate traineeships should be sent to the Chairman, Committee on Traineeships and Fellowships, University of Pennsylvania, School of Dentistry, 4001 Spruce Street, Philadelphia 4, Pennsylvania. Inquiries for the traineeships in periodontology should be sent to: Dean, Graduate School of Medicine, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Pennsylvania.

VETERANS ADMINISTRATION

The Veterans Administration Medical Teaching Group Hospital of Memphis, Tennessee announces that one residency in Prosthodontics and two rotating dental internships will be available for the year beginning July 1961. Information and application forms may be obtained from the Chief Dental Service, Veterans Administration Medical Teaching Group Hospital, Memphis, Tennessee.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

Tufts University announces three full-time graduate courses in Periodontology under the direction of Doctor Irving Glickman.

I. PERIODONTOLOGY I: This is a two-year (two academic years of eighteen consecutive months) specialist training program for individuals interested in qualifying for certification in Periodontia. It has been arranged in conformity with the requirements of the American Board of Periodontology. Approximately one-half of the course is devoted to didactic instruction and the remainder is clinical practice. In addition to training in the Periodontia clinic, the student is given the opportunity of participating in clinics of allied dental and medical specialties. This wide range of clinical contact provides a useful background for expert management of periodontal problems.

II. PERIODONTOLOGY II: This is a one-year specialist training program, is a modification of the two-year course and is offered for individuals with a year's previous graduate or postgraduate training in periodontology.

III. PERIODONTOLOGY: This program is intended for individuals with a primary interest in the academic and research aspects of Periodontology. It is an eighteen months course leading to the degree of Master of Science or Master of Dental Science. This course consists of the following: 1) didactic instruction in pre-clinical sciences with special emphasis on Periodontal pathology; 2) clinical training in Periodontology; 3) research and thesis. The facilities of the Ziskin Memorial Research Laboratory and other departments of the medical and dental schools are available for research activities. Upon the completion of such a program, one may prepare for specialty board certification by spending an additional six months in clinical study.

Tufts University School of Dental Medicine announces two fellowships intended for

graduate dentists interested in research and teaching careers in periodontology now are available. In addition to participation in an extensive research program, the fellowships offer training in periodontal pathology and an opportunity for preparing for advanced degrees. There is an annual stipend of $5,000, and the fellowships are renewable. Additional information and application may be obtained from Dr. Irving Glickman, Periodontology, 136 Harrison Avenue, Boston 11, Massachusetts.

BOSTON UNIVERSITY
MASSACHUSETTS MEMORIAL HOSPITALS

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University, and the Massachusetts Memorial Hospitals, announce the following postgraduate courses:

January 16-21, 1961. Periodontal Pathology by Drs. Henry M. Goldman and Jack Bloom. Fee $200.

April 24-29, 1961. Periodontal Therapy by Drs. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Jack Bloom, and Associates. Fee $200.

April 24-29, 1961. Advanced Course in Periodontal Therapy by Drs. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast and Associates. Fee $200.

May 22-26, 1961. Surgical Procedures in Periodontal Therapy by Dr. Lewis Fox and Associates. Fee $200.

The following week-end refresher course is also announced:

April 1, 1961. The Use of Ultrasonics in Periodontal Therapy by Dr. Henry M. Goldman of Brookline, Mass. Fee $35.

For further information write Director, Postgraduate Courses, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Avenue, Boston 18, Massachusetts.

NORTHWESTERN UNIVERSITY

Northwestern University Dental School announces a postgraduate course in Practical Periodontal Treatment, to be presented January 23, 24 and 25, 1961. Instructors will be Dr. Irving Glickman and Dr. Marjorie Houston. Tuition $150.00.

For further information write Director of Dental Postgraduate Study, Northwestern University Dental School, 311 East Chicago Avenue, Chicago 11, Illinois.

UNIVERSITY OF SOUTHERN CALIFORNIA

The University of Southern California School of Dentistry announces the following postgraduate courses:

February 12 and 13, 1961. Advanced Periodontal Therapy by Dr. Henry M. Goldman. Fee $60.00. Enrollment limited.

February 16, 23, March 2 and 9, 1961. Periodontics Review. Instructors, Robert L. Reeves, D.D.S., and Staff. Fee $50.00.

May 27 and 28, 1961. Periodontics. Saul Schluger, D.D.S. Fee $50.00. Enrollment limited.

Two semesters—Thursday mornings. Periodontics. Instructors, Robert L. Reeves, Aronald A. Ariaudo, Nathan Friedman, Daniel Feder, Leslie H. Levine and Sylvan Schireson. Academic credit not granted; certificate of completion is issued. Fee $550.00. Enrollment limited.

For further information write Division of Postgraduate Instruction, University of Southern California School of Dentistry, University Park, Los Angeles 7, California.

CHICAGO ACADEMY OF DENTAL RESEARCH

The Annual Seminar of the Chicago Academy of Dental Research will be held February 4 and 5, 1961 in the Conrad Hilton Hotel, Chicago, Illinois. The subject will be "Periodontia" and the clinician will be Dr. Henry M. Goldman. Fee, $125.

For further information write Dr. Sanford Schaffner, Director of Seminars, Chicago Academy of Dental Research, 10 North LaSalle Street, Chicago 2, Illinois.

WESTERN RESERVE UNIVERSITY

Western Reserve University School of Dentistry announces a postgraduate course in Periodontics, "Recent Developments in Periodontics and Occlusion" April 28, 29, 30, 1961. Dr. Sigurd Ramfjord is guest lecturer. Fee $100.00.

For further information write Director of Postgraduate Education, School of Dentistry, Western Reserve University, Cleveland 6, Ohio.

WESTERN SOCIETY OF PERIODONTOLOGY

The Western Society of Periodontology announces that the Eighth Annual Scientific Meeting will be held at the Tropicana Hotel in Las Vegas on March 19, 20, 21, 1961. The guest speaker will be Dr. Robert F. Moyers of the University of Michigan.

For further information, write Dr. Daniel Feder, 152 South Lasky Drive, Beverly Hills, California.

ARMED FORCES INSTITUTE OF PATHOLOGY

The Armed Forces Institute of Pathology will again present its five-day course "Pathology of the Oral Regions" April 10 to 14, 1961. This course will be available to civilians as well as military personnel.

For further information write Louis S. Hansen, Capt., DC, USN, Registrar, American Registry of Oral Pathology, Washington 25, D. C.

BICENTENNIAL OF THE DEATH OF PIERRE FAUCHARD TO BE RECOGNIZED

A committee to arrange a meeting honoring Dr. Pierre Fauchard which has been organized in France announces that the meeting will be held in Paris July 1 and 2, 1961. Anyone interested in participating in this program is urged to write Dr. M. Max Filderman, Secretary-General, Comite National du Bicentenaire de Fauchard, 67, rue de Tocqueville, Paris XVIIe, France.

MIDWEST SOCIETY OF PERIODONTOLOGY

The annual meeting of the Midwest Society of Periodontology will be held on February 5, at the Blackstone Hotel, in Chicago. Drs. Donald A. Kerr, Frank M. Wentz, and John R. Wilson are the essayists for the program. Registration may be made at the meeting; the fee for non-members is fifteen dollars.

TUFTS UNIVERSITY

Tufts University School of Dental Medicine will present "A Special Participation Course in Periodontology" April 24, 1961 through April 28, 1961. Tuition is $175; class is limited.

For further information write Dr. Irving Glickman, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Massachusetts.

1961 MEETING OF THE AMERICAN ACADEMY OF PERIODONTOLOGY

A communication from Dr. Sigurd Ramfjord urges anybody who has material they want to present at the 1961 meeting to contact Dr. Perry A. Ratcliff, 1998 "D" Street, San Bernardino, California, if it is in the form of an essay; Dr. Walter Cohen, 1913 Walnut Street, Philadelphia, Pennsylvania, if it is suitable for clinic, or Dr. Helmut Zander, 800 Main Street, East, Rochester, New York, if it is a research report.

HELP NEEDED!

There are many libraries throughout the world that would like to have back issues of the JOURNAL OF PERIODONTOLOGY, but we have no more of some of these issues in our office. We recently received a request from a library for the following:

Vol. 1	All issues	Vol. 14	No. 1
Vol. 2	All issues	Vol. 15	No. 1
Vol. 4	All issues	Vol. 18	No. 2
Vol. 5	All issues	Vol. 20	No. 2
Vol. 12	Nos. 1, 3, 4	Vol. 21	No. 1

Anyone wishing to dispose of their copies of these issues may send them directly to the library (the name may be obtained from the Editor) or they may be sent directly to the Editor, 1121 West Michigan Street, Indianapolis 2, Indiana.

UNIVERSITY OF TORONTO FACULTY OF DENTISTRY

The University of Toronto Faculty of Dentistry will present the following post-graduate courses of interest to periodontists:

Periodontics, May 8-12, 1961.

Further information about these courses may be obtained by writing the Chairman, Division of Postgraduate Studies, Faculty of Dentistry, University of Toronto, Toronto, Ontario, Canada.

MIDWEST SOCIETY OF PERIODONTOLOGY

The annual meeting of the Midwest Society of Periodontology will be held at the Blackstone Hotel, Chicago on Sunday February 5, 1961. Doctors Donald Kerr, Frank Wentz and John R. Wilson are the speakers of the Program. Non-members will be assessed a $15.00 registration fee.

Letters to the Editor . . . .

I would like to be permitted to say a few words in regard to the excellent and interesting paper, "Newer Concepts of Mucogingival Surgery," by Clifford Ochsenbein in the July, 1960, issue of the JOURNAL OF PERIODONTOLOGY.

The authorship of the repositioning operation is ascribed to A. A. Ariaudo and H. A. Tyrell in the JOURNAL OF PERIODONTOLOGY, 1957, when in fact this operation was suggested originally three years earlier by C. L. Nabers, also in the JOURNAL OF PERIODONTOLOGY.

The second point that I wish to make is the following: In Dr. Ochsenbein's paper, credit is given for development of certain procedures and operations to some highly respected members of our specialty; however, this credit is given by means of references to "personal communication" *with no date*. To the knowledge of the writer of this letter it is not customary in scientific writing and among scientists to base credit on such evidence, but rather on publications in scientific journals or, rather rarely, on presentation before a recognized group at a stated meeting and date. A reference to "personal communication" *with date* is only used where the work referred to is of recent origin, or to corroborate relatively minor points as, for instance, similar cases seen by a colleague. There is good reason for this custom. If a scientist considers a piece of his work, be it an investigation or a procedure, as sufficiently important, he is expected to publish it at an early time so that the rest of his confreres may benefit from his findings.

In conclusion, let me say that I have high regard for Dr. Ochsenbein and his accomplishments and also for the men he refers to as the originators of the operations and techniques mentioned. The scientist produces in order to help mankind and to advance knowledge in his chosen field; still it would be helpful, I believe, if we in periodontology would follow the rules set by older and better established disciplines in the matter of credits.

Yours sincerely,
Frank G. Everett, M.S., D.M.D., M.D.

I believe we are grossly underestimating the intelligence of the average American by not being willing to stress the importance of proper home care for total mouth health—teeth *and* supporting tissues. Could not your influence be used to help some group scientifically evaluate the value of using dental floss and brushes soft enough to brush "into the gums"? Have we really *proven* that it is best to brush "away from the gums"? This is so imminently tied in with work in the field of preventive dentistry. Teeth and the supporting structures can be separated when one enters a research laboratory but they *cannot* be separated when you try to tell the general public in a very few words how to care for the teeth at home.

I have nothing but clinical evidence to support this claim but I have *seen* what happens in the mouths of my patients when they start cleaning out the gingival crevices by brushing "into the gums" instead of "away from" and I have *seen* what happens when they started using dental floss regularly to get down in that area where *no* brush can reach. Colleagues who teach this method tell me the same thing. I would be foolish to imply that *all* my patients follow these instructions but *most* of them do and I have an average small town practice in a typical community.

Many of these patients say they have been brushing their teeth like that all the time—that they never could use the 'up and down' method anyway. So what they have been doing, of course, is using a hard brush horizontally and accomplishing little but gum recession and erosion instead of using a soft brush at a 45 degree angle to the teeth and scrubbing back and forth.

The very fact that toothbrush manufacturers themselves come out so often with such outlandish styles, handles and bristles is evidence that they are just groping around in the dark with no one to direct them. Could not we help some one prove there is a better toothbrush and a correct way to use it?

James W. Thomas
101 N. Trigg Ave., Gallatin, Tennessee

First of all, I wish to express my gratitude for your abstract of my article "Study of Periodontal Disease and Occlusal Trauma by Use of the Index of Alveolar Resorption," published in THE JOURNAL OF PERIODONTOLOGY 1960, July issue.

I have been asked to explain the method of application of alveolar resorption (IAR). The available applications of IAR can be noted in our articles.[2,3,4,5,6,7,8] We found that the correlation between tooth mobility (TM) and IAR to the relative severity of periodontal disease of the single rooted teeth can be calculated according to the following formula:[5]

$$\frac{IAR}{0.023} + \frac{}{\text{the average value of physiological TM}} = \text{TM of the relative moderate severity of periodontal,}$$

disease, named as "comparing factor" (CF).

(The average physiologic value of TM, i.e., the TM of the healthy periodontium are according to Muehlemann:[1] for incisors—12, for canines—8, for first lower premolars—10, for second lower and upper premolars—9).

There is no significant deviation from the respective formula up to the value of about 0.200 for IAR. Over that value the deviation is probably unproportionally increased.

The CF represents the determinated value of TM (of moderate severity of periodontal disease) related to determined bone resorption, express by means of IAR. If, however, a greater value for TM exists than the CF suggests, this means the periodontal process is increased. If, on the other hand, the TM shows the lower value than the CF, the periodontal disease process is decreased.

For study of periodontal disease and occlusal trauma we prepared a table for comparing of the examined values of TM with the values of CF and IAR,[6] which quite simplifies this procedure.

We will explain any further details concerning IAR upon request.

Thank you very much for your attention.

Sincerely yours,
Dr. Bozidar Kuljaca

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